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TRANSACTIONS

OF THE

AMERICAN SURGICAL ASSOCIATION

ROYAL SOCIETY OF MEDICINE
JUN 24
1927

VOLUME THE FORTY-FOURTH

EDITED BY

JOHN H. JOPSON, M.D.
RECORDER OF THE ASSOCIATION

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1926

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ROYAL SOCIETY OF MEDICINE
JUN 24
1927

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NOTICE

This volume of TRANSACTIONS contains the papers read before the Association at the Meeting held May 24, 25 and 26, 1926.

The Association assumes no responsibility for the statements and opinions published in this volume.

ROYAL SOCIETY OF MEDICINE
JUN 24
1927

OFFICERS OF THE ASSOCIATION

ELECTED MAY, 1926

PRESIDENT

HARVEY CUSHING, M.D.

VICE-PRESIDENTS

ALEXANDER PRIMROSE, M.D.
ARTHUR AYER LAW, M.D.

SECRETARY

ROBERT B. GREENOUGH, M.D.,
8 Marlborough Street, Boston, Mass.

TREASURER

EUGENE H. POOL, M.D.,
107 East Sixtieth Street, New York City.

RECORDER

JOHN H. JOPSON, M.D.,
1824 Pine Street, Philadelphia, Pa.

COUNCIL

GEORGE W. CRILE, M.D.
GEORGE E. BREWER, M.D.
JOHN H. GIBBON, M.D.
HARVEY CUSHING, M.D.
ROBERT B. GREENOUGH, M.D.
EUGENE H. POOL, M.D.
JOHN H. JOPSON, M.D.
} *Ex-officio.*

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RECORDER, SECRETARY AND TREASURER, *ex-officio.*

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PRESIDENT, SECRETARY, RECORDER AND THE CHAIRMAN OF THE ADJUNCT LOCAL COMMITTEE OF ARRANGEMENTS, *ex-officio.*

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(Report by Title)

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ASTLEY P. C. ASHHURST, M.D.
LINCOLN DAVIS, M.D.

Place of next meeting:

RICHMOND, VA.

ADJUNCT LOCAL COMMITTEE OF ARRANGEMENTS

STUART McGUIRE, M.D.
J. SHELTON HORSLEY, M.D.

DELEGATE TO TRIENNIAL CONGRESS IN WASHINGTON

WILLIAM H. CARMALT, M.D.
Alternate, JAMES F. MITCHELL, M.D.

LIST OF THE PRESIDENTS

OF THE

ASSOCIATION FROM THE TIME OF ITS INSTITUTION

‡ Deceased.

ELECTED

1880. ‡SAMUEL DAVID GROSS, M.D., LL.D., D.C.L. (Oxon.), LL.D. (Cantab.), LL.D. (Edin.)—Founder of the Association.
1883. ‡EDWARD MOTT MOORE, M.D., LL.D.
1884. ‡WILLIAM THOMPSON BRIGGS, M.D.
1885. ‡MOSES GUNN, A.M., M.D., LL.D.
1886. ‡HUNTER McGUIRE, M.D., LL.D.
1887. ‡D. HAYES AGNEW, M.D., LL.D.
1888. ‡DAVID WILLIAMS CHEEVER, M.D., LL.D.
1889. ‡DAVID W. YANDELL, M.D.
1890. ‡CLAUDIUS HENRY MASTIN, M.D., LL.D.
1891. ‡PHINEAS SANBORN CONNER, M.D., LL.D.
1892. ‡NICHOLAS SENN, M.D., Ph.D., LL.D.
1893. ‡JAMES EWING MEARS, A.M., M.D., LL.D.
1894. FREDERIC SHEPARD DENNIS, M.D.
1895. ‡LOUIS McLANE TIFFANY, A.M. (Cantab.), M.D.
1896. JOHN COLLINS WARREN, M.D., LL.D. (Jefferson), Hon. F.R.C.S. (Eng.).
1897. ‡THEODORE F. PREWITT, M.D.
1898. WILLIAM W. KEEN, A.M., M.D., LL.D., Hon. F.R.C.S. (Eng., Edin. and Ireland), Hon. F.A.C.S.
1899. ROBERT F. WEIR, A.M., M.D., Hon. F.R.C.S. (Eng.).
1900. ‡ROSWELL PARK, M.D.
1901. ‡DE FOREST WILLARD, A.M., M.D., Ph.D.
1902. ‡MAURICE H. RICHARDSON, A.B., M.D.
1903. ‡NATHANIEL P. DANDRIDGE, M.D.
1904. ‡GEORGE BEN JOHNSTON, M.D.
1905. ALBERT VANDER VEER, M.D.
1906. ‡DUDLEY P. ALLEN, M.D., LL.D.
1907. WILLIAM H. CARMALT, M.D.
1908. ‡C. B. G. de NANCRÈDE, A.M., M.D., LL.D.
1909. RUDOLPH MATAS, M.D.

ELECTED

1910. ‡RICHARD H. HARTE, M.D.
1911. ‡ARPAD G. GERSTER, M.D., CHIR.D.
1912. ‡CHARLES A. POWERS, A.M., M.D.
1913. WILLIAM J. MAYO, A.M., M.D., D.Sc., F.R.C.S. (EDIN.), LL.D. (TOR.).
1914. GEORGE E. ARMSTRONG, C.M., M.D.
1915. ‡ROBERT G. LE CONTE, A.B., M.D.
1916. ‡SAMUEL J. MIXTER, S.B., M.D.
1917. THOMAS W. HUNTINGTON, A.B., M.D., LL.D.
1918. LEWIS S. PILCHER, A.M., M.D., LL.D.
1919. GEORGE E. BREWER, M.D.
1920. ‡JOHN B. ROBERTS, M.D.
1921. JOHN M. T. FINNEY, M.D.
1922. L. L. McARTHUR, M.D.
1923. GEORGE W. CRILE, M.D.
1924. ‡ALBERT J. OCHSNER, B.S., M.D., F.R.M.S. (ENG. and IRE.), LL.D.
1925. JOHN H. GIBBON, M.D.
1926. HARVEY CUSHING, A.M., M.D., ScD., LL.D., F.R.C.S.

ROYAL SOCIETY OF MEDICINE
JUN 24 1927

FELLOWS

OF THE

AMERICAN SURGICAL ASSOCIATION

MAY, 1926

* Denotes Original Fellows.
† Denotes Senior Fellows.
‡ Denotes Deceased Fellows.

ELECTED

†1890. Abbe, Robert, M.D., 40 W. Fifty-ninth Street, New York. Senior Surgeon to St. Luke's Hospital. *Vice-President*, 1901. *Member of Council*, 1908.

‡1902. Andrews, E. Wyllys, A.M., M.D., 122 S. Michigan Avenue, Chicago, Ill. Professor of Surgery, Northwestern University Medical School; Surgeon to St. Luke's Hospital and Michael Reese Hospital; Surgeon-in-Chief to Cook County Hospital; Consulting Surgeon to the Provident Hospital. *Vice-President*, 1923.

1916. Archibald, Edward William, B.A., M.D., C.M., 52 Westmount Boulevard, Montreal, Canada. Professor of Surgery and Director of the Surgical Department, McGill University; Surgeon, Royal Victoria Hospital; Associate Surgeon, Children's Memorial Hospital. *Vice-President*, 1925.

†1901. Armstrong, George E., M.D., C.M., C.M.G., LL.D. (Queens), D.Sc. (Liverpool), M.Ch. (Dublin), 320 Mountain Street, Montreal, Canada. Emeritus Professor of Surgery and of Clinical Surgery, McGill University; Consulting Surgeon to the Royal Victoria Hospital, the Montreal General Hospital, the Western Hospital and to the Protestant Hospital for the Insane, Verdun; Colonel, C.A.M.C. *President*, 1914.

ELECTED

1913. ASHHURST, ASTLEY PASTON COOPER, A.B., M.D., N. E. Corner Sixteenth and Spruce Streets, Philadelphia. Professor of Clinical Surgery in the University of Pennsylvania; Surgeon to the Episcopal Hospital; Surgeon to the Philadelphia Orthopædic Hospital and Infirmary for Nervous Diseases; Colonel, Medical Reserve Corps, U. S. Army.

1922. BAILEY, FRED WARREN, B.Sc., M.D., 611 Metropolitan Building, St. Louis, Mo. Associate Professor of Surgery, St. Louis University School of Medicine; Surgeon to St. John's Hospital; Visiting Surgeon, St. Louis City Hospital; Surgeon and Chief of Staff, Shriners' Hospital.

1920. BALCH, FRANKLIN G., A.M., M.D., F.A.C.S., 279 Clarendon Street, Boston, Mass. Chief of East Surgical Service, Massachusetts General Hospital; Surgeon at the Faulkner Hospital; Associate in Surgery, Graduate Courses in Medicine, Harvard Medical School.

1917. BALFOUR, DONALD CHURCH, M.B., M.D., Rochester, Minn. Professor of Surgery, University of Minnesota; Chief of Surgery, Mayo Foundation; Surgeon to St. Mary's Hospital, Rochester. *Vice-President*, 1922.

1925. BANCROFT, FREDERIC W., A.B., M.D., 100 East Sixty-sixth Street, New York, N. Y. Associate in Surgery, College of Physicians and Surgeons, Columbia University; Surgical Director, Lincoln Hospital; Consulting Surgeon, U. S. Veteran's Hospital, No. 81; formerly Major, Medical Corps, U. S. Army.

†1906. BARROW, DAVID, M.D., 190 N. Upper Street, Lexington, Ky. Surgeon to St. Joseph and Good Samaritan Hospitals; recently Lieut.-Col., M.C.; Commanding Officer, Base Hospital 40.

‡1882. BARTON, JAMES M., A.M., M.D., 32 North New Hampshire Avenue, Atlantic City, N. J. (Retired.) Formerly Professor of Clinical Surgery, Jefferson Medical College; and Surgeon to the Philadelphia and German (now Lankenau) Hospitals.

1926. BAZIN, ALFRED T., M.D., C.M., Medical Arts Building, Montreal, Canada. Professor of Surgery and Clinical Surgery, McGill University; Attending Surgeon, Montreal General Hospital; Consulting Surgeon, Champlain Valley Hospital, Plattsburg, N. Y.

1924. BEER, EDWIN, A.B., M.D., 45 East Eighty-fifth Street, New York City. Attending Surgeon, Bellevue Hospital, 4th Division, and at Mt. Sinai Hospital.

ELECTED

1915. Besley, Frederic A., M.D., F.A.C.S., 215 North Sheridan Road, Waukegan, Ill. Professor of Surgery, Northwestern University Medical School; Attending Surgeon, Victory Memorial Hospital, Waukegan, and Cook County Hospital, Chicago, Ill.

1900. Bevan, Arthur Dean, M.D., 122 S. Michigan Boulevard, Chicago. Professor and Head of the Department of Surgery, Rush Medical College, University of Chicago; Surgeon to Presbyterian Hospital.

†1901. Binnie, John Fairbairn, M.A., M.B., C.M. (Aberdeen), 917 Rialto Building, Kansas City, Mo. *Vice-President*, 1907 and 1919.

1918. Blair, Vilray Papin, M.D., Metropolitan Building, St. Louis, Mo. Professor of Oral Surgery, Washington University Dental School; Associate Professor of Clinical Surgery, Washington University Medical School; Visiting Surgeon, St. Luke's Hospital; Associate Surgeon to St. Louis Maternity and Mullanphy Hospital, St. Louis Children's Hospital, and Barnes Hospital.

†1906. Blake, John Bapst, A.M., M.D., 311 Beacon Street, Boston, Mass. Assistant Professor in Surgery, Harvard Medical School; Surgeon-in-Chief to the Boston City Hospital; Consulting Surgeon to Long Island Hospital; Consulting Surgeon to Boston Insane Hospital, and to the Reformatory for Women, Framingham, Mass.

†1902. Blake, Joseph A., A.B., Ph. B., M.A. (Hon.), M.D., F.A.C.S., 116 East Fifty-third Street, New York. International Surgical Society; American Association of Anatomists; Corresponding Member, French Surgical Society; New York Academy of Medicine. Surgical Director, Reconstruction Hospital; Consulting Surgeon, Roosevelt, St. Luke's, Lincoln, St. John's (Yonkers), Tarrytown and Dobbs Ferry Hospitals.

1901. Bloodgood, Joseph Colt, B.S. (Wis., 1888), M.D. (Penna., 1891); 904 N. Charles Street, Baltimore, Md.; residence, 18 Midvale Road, Roland Park, Md. Associate Professor of Clinical Surgery, Johns Hopkins University; Surgeon to St. Agnes's Hospital, Baltimore. *Vice-President*, 1924.

†1900. Brewer, George Emerson, M.D., LL.D., F.A.C.S., F.R.C.S. (Ireland), *Member of Council*, 19 East Sixty-fifth Street, New York City. Emeritus Professor of Surgery in College of Physicians and Surgeons, Columbia University; Consulting Surgeon to the Presbyterian Hospital, Roosevelt Hospital,

ELECTED

City Hospital, Woman's Hospital, St. Vincent's Hospital, Knickerbocker Hospital, Fifth Avenue Hospital, Manhattan Eye, Ear, Nose and Throat Hospital, Ophthalmic and Aural Institute, St. Bartholomew's Hospital and Clinic, and House of the Holy Comforter, New York; the Flushing Hospital, Perth Amboy Hospital, the Muhlenberg Hospital of Plainfield, N. J., and Christ Hospital, Jersey City. *Vice-President*, 1913; *President*, 1919. *Member of Council*, 1920–23.

1917. BREWSTER, GEORGE W. W., A.B., M.D., F.A.C.S., 213 Beacon Street, Boston, Mass. Associate in Surgery, Harvard Graduate School; Visiting Surgeon, Massachusetts General Hospital; Consulting Surgeon to Frost Hospital, Chelsea, Mass.; Leonard Morse Hospital, Natick, Mass.; Milford Hospital, Milford, Mass.; Brockton Hospital, Brockton, Mass.

†1915. BRINSMADE, W. B., A.B., M.D., 166 Columbus Heights, Brooklyn, N. Y. Consulting Surgeon to St. Christopher's Hospital; Senior Surgeon, Brooklyn Hospital; Commander, U.S.N.R.F., M.C.

1925. BROOKS, BARNEY, B.S., M.D., Vanderbilt University Hospital, Nashville, Tenn. Professor of Surgery, Vanderbilt University Medical School; Surgeon-in-Chief, Vanderbilt University Hospital.

1911. BRUCE, HERBERT A., M.D. (Toronto Univ.), L.R.C.P. (London), F.R.C.S. (England), 64 Bloor Street East, Toronto, Canada. Professor of Clinical Surgery, University of Toronto; Consulting Surgeon to the Toronto General Hospital; Colonel, Canadian Army Medical Corps and Royal Army Medical Corps; Consulting Surgeon to the British Armies in France, 1917–18.

1911. BUCHANAN, J. J., A.M., M.D., PH.D., 1409 N. Highland Avenue, Pittsburgh, Pa. Professor of Surgery, University of Pittsburgh; Surgeon to Mercy Hospital.

†1900. BUNTS, FRANK E., M.D., Euclid Avenue and Ninety-third Street, Cleveland, Ohio. Professor of Principles of Surgery and Clinical Surgery in Medical College of Western Reserve University; Visiting Surgeon and Chief of Staff to St. Vincent's Charity Hospital.

1924. CABOT, HUGH, A.B., M.D., University Hospital, Ann Arbor, Mich. Professor of Surgery, University of Michigan; Surgeon-in-Chief, University Hospital.

ELECTED

‡1921. CAMPBELL, WILLIAM F., M.D., 394 Clinton Avenue, Brooklyn, N. Y. Surgeon-in-Chief to the Trinity Hospital.

†1885. CARMALT, WILLIAM H., A.M., M.D., 261 St. Ronan Street, New Haven, Conn. Emeritus Professor of Surgery in Yale University; Vice-President and Consulting Surgeon to the New Haven Hospital. *President*, 1907; *Vice-President*, 1895; *Member of Council*, 1908.

1909. CARREL, ALEXIS, L.B., SC.B., M.D., Rockefeller Institute for Medical Research, Sixty-sixth Street and Avenue A, New York, N. Y. *Vice-President*, 1919.

†1896. CARSON, NORMAN BRUCE, M.D., 7006 Maryland Drive, St. Louis, Mo. Surgeon to St. Louis and Mullanphy Hospitals and to the St. Vincent Insane Asylum, etc. *Vice-President*, 1903.

1922. CHEEVER, DAVID, A.B., M.D., Peter Bent Brigham Hospital, Boston, Mass. Associate Professor of Surgery, Harvard Medical School; Surgeon to the Peter Bent Brigham Hospital.

1926. CLARK, EDMUND D., M.D., Hume-Mansur Building, Indianapolis, Ind. Professor of Surgery, Indiana University School of Medicine; Surgeon to the Robert W. Long, Methodist and City Hospitals; Colonel, Medical Reserve Corps, U. S. A.

1920. CLINTON, MARSHALL, M.D., 556 Franklin Street, Buffalo, New York. Associate Professor of Surgery, University of Buffalo, Department of Medicine; Professor of Oral Surgery, University of Buffalo, Dental Department; Attending Surgeon, Buffalo General, Erie County, and Buffalo City Hospitals.

1920. CLOPTON, MALVERN BRYAN, M.D., 405 Humboldt Building, St. Louis, Mo. Associate in Surgery, Washington University Medical School; Visiting Surgeon, St. Luke's Hospital; Assistant Surgeon, Barnes Hospital; Associate Surgeon, St. Louis Children's Hospital.

†1916. CODMAN, ERNEST AMORY, A.B., M.D., 227 Beacon Street, Boston, Mass.

†1898. COLEY, WILLIAM B., M.D., The Medical Chambers, 114 East Fifty-fourth Street, New York. Attending Surgeon to the Ruptured and Crippled and the Memorial Hospitals; Consulting Surgeon to the Fifth Avenue, the Mary McClellan (Cambridge, N. Y.), and the Sharon (Sharon, Conn.) Hospitals; Professor of Clinical Cancer Research at Memorial Hospital, Cornell University Medical College.

ELECTED

1922. Connors, John Fox, A.M., M.D., 59 East Fifty-fourth Street, New York City. Surgical Director, Harlem Hospital, New York City; Consulting Surgeon, St. Mary's Hospital, Waterbury, Conn.

1914. Cotton, Frederick J., A.B., M.D., A.M., 520 Commonwealth Avenue, Boston, Mass. Visiting Surgeon to the Boston City Hospital; Surgeon-in-Chief, Beth Israel Hospital; Associate in Surgery, Harvard Medical School; Consultant in Surgery, United States Public Health Service, Northeastern District; also Consultant in Surgery, New England Hospital for Women and Children; Consultant in Surgery, Peterboro Hospital.

†1905. Crile, George W., M.D., *Member of Council*, Euclid Avenue and Ninety-third Street, Cleveland, Ohio. Surgeon, Cleveland Clinic Hospital. *President*, 1923.

1906. Cushing, Harvey, A.M., M.D., Sc.D., LL.D., F.R.C.S., *President*, 721 Huntington Avenue, Boston. Professor of Surgery, Harvard University.

†1896. Cushing, Hayward Warren, A.B., M.D., 70 Commonwealth Avenue, Boston, Mass. Consulting Surgeon, Boston City Hospital.

1925. Cutler, Elliott Carr, A.B., M.D., F.A.C.S., D.S.M. (U.S.A.), Lakeside Hospital, Cleveland, Ohio. Professor of Surgery, Western Reserve University; Director of Surgery, Lakeside Hospital.

†1897. Da Costa, John Chalmers, M.D., 2045 Walnut Street, Philadelphia. Samuel D. Gross Professor of Surgery, Jefferson Medical College; Surgeon to the Hospital of the Jefferson Medical College; Consulting Surgeon to the Philadelphia General Hospital, Saint Joseph's Hospital and Misericordia Hospital.

1925. Dandy, Walter Edward, A.B., A.M., M.D., The Johns Hopkins Hospital, Baltimore, Md. Associate Professor of Surgery, Johns Hopkins University, in charge of Neurological Surgery.

1917. Darrach, William, A.B., M.A., M.D., 437 West Fifty-ninth Street, New York City. Associate Professor of Surgery and Dean of the Medical Faculty, College of Physicians and Surgeons, Columbia University; Consulting Surgeon, Presbyterian Hospital, New York City; Greenwich Hospital, Greenwich, Connecticut; St. John's Hospital, Yonkers, N.Y., and Memorial Hospital, Morristown, N. J.

ELECTED

1923. DAVID, VERNON C., A.B., M.D., 25 East Washington Street, (Room 1927), Chicago, Ill. Assistant Professor of Surgery, Rush Medical College; Associate Attending Staff, Surgery, Presbyterian Hospital; Attending Staff, Surgery, Cook County Hospital; Senior Attending Staff, Surgery, Evanston Hospital.

1916. DAVIS, CARL, B., A.B., M.D., 122 South Michigan Boulevard, Chicago, Ill. Associate Professor of Clinical Surgery, Rush Medical College; Associate Surgeon, Presbyterian Hospital, Chicago.

1917. DAVIS, JOHN STAIGE, PH.B., M.D., F.A.C.S., Severn Apartments, Baltimore, Md. Associate Professor of Clinical Surgery, Johns Hopkins University; Visiting and Plastic Surgeon, Union Memorial Hospital, Hospital for the Women of Maryland, Children's Hospital School; Assistant Visiting Surgeon and Dispensary Surgeon, Johns Hopkins Hospital.

1919. DAVIS, LINCOLN, A.B., M.D., 205 Beacon Street, Boston, Mass. Associate in Surgery, Harvard Graduate School of Medicine; Visiting Surgeon, Massachusetts General Hospital; Consulting Surgeon, Heywood Memorial Hospital, Gardner; Milford Hospital, Milford.

†1912. DEAVER, HARRY C., M.D., 337 South Eighteenth Street, Philadelphia. Surgeon-in-Chief to Kensington Hospital for Women; Surgeon to the Episcopal Hospital and to the Children's Hospital of the Mary Drexel Home.

1892. DEAVER, JOHN B., M.D., SC.D., LL.D., 1830 De Lancey Street, Philadelphia. Emeritus John Rhea Barton Professor of Surgery, University of Pennsylvania, Philadelphia; Surgeon-in-Chief to the Lankenau Hospital, Philadelphia.

1910. DELATOUR, H. BEECKMAN, M.D., 73 Eighth Avenue, Brooklyn, N. Y. Attending Surgeon, St. John's and Norwegian Hospitals; Consulting Surgeon, Kingston Avenue Hospital, Brooklyn; St. Mary's Hospital, Jamaica; Northport Hospital, and South Side Hospital, Bay Shore.

†1882. DENNIS, FREDERIC SHEPARD, M.D., F.R.C.S. (Eng.), 1136 Fifth Avenue, New York. Professor Emeritus of Clinical Surgery, Cornell University, New York City; Consulting Surgeon to Bellevue and St. Vincent's Hospitals; St. Joseph's Hospital, Yonkers, and Montefiore Home, New York City; Litchfield County Hospital, Winsted, Conn. *President*, 1894; *Vice-President*, 1887; *Member of Council*, 1889.

1924. DOUGLASS, JOHN, SC.B., M.D., 568 Park Avenue, New York City. Clinical Professor of Surgery, New York University and Bellevue Medical College; Attending Surgeon, Bellevue

Hospital; Associate Surgeon, St. Luke's Hospital; Surgical Director, Knickerbocker Hospital.

†1910. Dowd, Charles N., A.M., M.D., Sc.D., 589 Park Avenue, New York, N. Y. Professor of Clinical Surgery, Columbia University; Consulting Surgeon, Roosevelt Hospital, Memorial Hospital, St. Mary's Free Hospital for Children, New York, Richmond Memorial Hospital, and Lulu Thorley Home for Crippled Children. *Member of Council*, 1918.

1914. Downes, William A., M.D., 424 Park Avenue, New York City. Professor of Clinical Surgery, Columbia University, College of Physicians and Surgeons; Attending Surgeon, St. Luke's and Babies' Hospitals; Consulting Surgeon, Memorial, Stamford Hospital, Stamford, Conn., Manhattan State Hospital, Hospital for Ruptured and Crippled, Infirmary for Women and Children, New York, and United Hospital, Portchester, N. Y.

1924. Drennen, Earle, A.B., M.D., 519 American Trust Building, Birmingham, Alabama. Attending Surgeon, Hillman Hospital, St. Vincent's Hospital and Children's Hospital.

1919. Eastman, Joseph Rilus, A.M., B.Sc., M.D., F.A.C.S., 331 North Delaware Street, Indianapolis, Ind. Professor of Surgery, Indiana University School of Medicine; Visiting Surgeon to City Hospital.

1925. Eberts, Edward Melchior, M.D., M.R.C.S. (Eng.), L.R.C.P. (Lond.), F.A.C.S., 219 Peel Street, Montreal, Canada. Associate Professor of Surgery and Clinical Surgery, McGill University; Attending Surgeon, Montreal General Hospital.

1926. Eliason, E. L., A.B., M.D., Sc.D., F.A.C.S., 326 South Nineteenth Street, Philadelphia, Pa. Professor of Clinical Surgery, University of Pennsylvania; Assistant Professor of Surgery, The Graduate School of Medicine, University of Pennsylvania; Surgeon to University and Howard Hospitals; Assistant Surgeon to Philadelphia General Hospital; Consulting Surgeon to Lebanon Sanitarium and Burlington County Hospital.

1901. Eliot, Ellsworth, Jr., M.D., 34 East Sixty-seventh Street, New York. Surgeon to the Polyclinic Hospital; Consulting Surgeon to the Grosveneur Hospital, N. Y.; Lawrence Hospital, Bruneville, N. Y.; Nasson Hospital, Mineola, L. I.; White Plains Hospital, White Plains, N. Y.; Hackensack Hospital, Hackensack, N. J.; Mountainside Hospital, Montclair, N. J.; Skin and Cancer Hospital, New York and Northeastern Dispensary, New York. *Vice-President*, 1922.

ELECTED

1911. Elting, Arthur W., A.B., M.D., LL.D., 119 Washington Avenue, Albany, N. Y. Professor of Surgery in the Albany Medical College; Surgeon to the Albany Hospital and to the Child's Hospital, Albany, N. Y.

1926. Erdman, Seward, A.B., M.D., 163 East Sixty-fifth Street, New York, N. Y. Associate Professor of Clinical Surgery, College of Physicians and Surgeons, Columbia University; Associate Attending Surgeon, New York Hospital; Consulting Surgeon, Memorial Hospital, Morristown, N. J.

†1896. Estes, William Lawrence, A.M., M.D., Bethlehem, Pa. Emeritus Surgeon-in-Chief of St. Luke's Hospital.

†1898. Eve, Duncan, A.M., M.D., Eve Building, Nashville, Tenn. Head of the Department and Professor of Surgery and Clinical Surgery, Medical Department, Vanderbilt University; Chief Surgeon of the Louisville & Nashville Railroad; Chief Surgeon of the N. C. & St. Louis Railroad; Attending Surgeon to St. Thomas' Hospital; Consulting Surgeon to Nashville City and Woman's Hospitals.

†1899. Finney, John M. T., M.D., 1300 Eutaw Place, Baltimore, Md. Professor of Clinical Surgery, Johns Hopkins University; Surgeon-in-Chief to Johns Hopkins Hospital. *President*, 1921. *Member of Council*, 1922–24.

1921. Fisher, William A., M.D., 715 Park Avenue, Baltimore, Md. Associate in Clinical Surgery, Johns Hopkins University; Assistant Visiting Surgeon, Johns Hopkins Hospital; Visiting Surgeon, Union Memorial Hospital, Church Home and Infirmary, Hospital for the Women of Maryland, Children's Hospital School.

1904. Frazier, Charles Harrison, M.D., Sc.D., 3600 Walnut Street, Philadelphia, Pa. John Rhea Barton Professor of Surgery, University of Pennsylvania; Surgeon-in-Chief to the University Hospital.

†1898. Freeman, Leonard, B.S., M.A., M.D., 2611 East Seventh Avenue, Denver, Col. Professor of Surgery, Medical Department, University of Colorado; Surgeon to St. Joseph's Hospital; Consulting Surgeon to Children's, St. Luke's, Beth Israel and National Jewish Hospitals, and to the Denver General Hospital, Denver, Col. *Vice-President*, 1908.

†1910. Gage, Homer, A.M., M.D., 72 Pearl Street, Worcester, Mass. Surgeon-in-Chief to Memorial Hospital; Consulting Surgeon

ELECTED

to Worcester City and St. Vincent's Hospitals. *Vice-President*, 1923.

1922. GALLIE, WILLIAM E., M.D., F.A.C.S., F.R.C.S. (Eng.), 143 College Street, Toronto, Canada. Hunterian Professor, Royal College of Surgeons of England; Associate Professor of Surgery, University of Toronto; Surgeon-inChief, Hospital for Sick Children, Toronto.

1919. GAUB, OTTO CARL, M.D., F.A.C.S. Hotel Schenley, Pittsburgh, Pa. Surgeon to Allegheny General Hospital and to Columbia Hospital.

†1882. GAY, GEORGE WASHINGTON, A.M., M.D., Chestnut Hill, Mass. Senior Surgeon to the Boston City Hospital. *Member of Council*, 1882.

1906. GIBBON, JOHN H., M.D., *Member of Council*, 1608 Spruce Street, Philadelphia. Professor of Surgery, Jefferson Medical College and Surgeon to the Jefferson College Hospital; Surgeon to the Pennsylvania Hospital and Consulting Surgeon to the Bryn Mawr Hospital. *Secretary*, 1915–1922. *President*, 1925.

†1906. GIBSON, C. L., M.D., 72 East Fifty-fourth Street, New York, N. Y. Professor of Surgery, Cornell University Medical College; Attending Surgeon to the First Surgical (Cornell) Division of the New York Hospital. Membre Associé-étranger Société Nationale de Chirurgie de Paris; Consulting Surgeon to St. Luke's, City, and General Memorial Hospitals; New York State Hospital for Care of Crippled and Deformed Children; South Side Hospital, Bay Shore; Vassar Brothers Hospital, Poughkeepsie. *Vice-President*, 1915.

1920. GRAHAM, EVARTS A., A.B., M.D., Euclid Avenue and Kingshighway, St. Louis, Mo. Professor of Surgery, Washington University Medical School, St. Louis, Missouri; Surgeon-in-Chief to Barnes Hospital and St. Louis Children's Hospital.

1911. GREENOUGH, ROBERT B., A.B. (Harvard), M.D. (Harvard), *Secretary*, 8 Marlborough Street, Boston, Mass. Assistant Professor of Surgery, Harvard Medical School; Visiting Surgeon, Massachusetts General Hospital; Director of Cancer Commission, Harvard University.

1922. GUERRY, LE GRAND, M.D., F.A.C.S., D.C.L., 1831 Pendleton Street, Columbia, S. C. Surgeon to Columbia Hospital, Columbia, S. C.

1924. GUTHRIE, DONALD, PH.B., M.D., Sayre, Pa. Surgeon-in-Chief, Robert Packer Hospital.

ELECTED

1916. HAGGARD, WILLIAM D., M.D., F.A.C.S., 706 Church Street, Nashville, Tenn. Professor of Surgery, Vanderbilt University; Surgeon to St. Thomas' Hospital.

‡1909. HALSTEAD, ALBERT E., M.D., 30 N. Michigan Avenue, Chicago, Ill. Professor of Surgery, School of Medicine, University of Illinois; Attending Surgeon to the St. Luke's Hospital; Consulting Surgeon to the Illinois Charitable Eye and Ear Infirmary.

1909. HAMANN, CARL A., M.D., 416 Osborn Building., Cleveland, Ohio. Professor of Applied Anatomy and Clinical Surgery, Western Reserve University; Visiting Surgeon to Charity and City Hospitals.

1900. HARRIS, MALCOLM L., M.D., 25 E. Washington Street, Chicago, Ill. Professor of Surgery, Chicago Policlinic; Attending Surgeon, Alexian Brothers' Hospital, Henrotin Hospital, Passavant Hospital.

1916. HARTWELL, JOHN A., PH.B., M.D., 27 East Sixty-third Street, New York. Associate Professor of Surgery and Professor of Clinical Surgery, Cornell University Medical College; Director of Surgery, Cornell University Division, Bellevue Hospital; Consulting Surgeon to Memorial Hospital, Reconstruction Hospital, New York Infirmary for Women and Children, Lincoln Hospital, Lawrence Hospital, Bronxville, N. Y., and United Hospital, Portchester, N. Y.

1926. HARVEY, SAMUEL C., PH.B., M.D., New Haven Hospital, New Haven, Conn. Surgeon-in-Chief, New Haven Hospital and New Haven Dispensary; Consulting Surgeon, Meriden Hospital, Meriden, Conn.

1922. HEUER, GEORGE J., M.D., Cincinnati General Hospital, Cincinnati, Ohio. Professor of Surgery, University of Cincinnati College of Medicine; Surgical Director of the Cincinnati General Hospital.

1922. HITZROT, JAMES MORLEY, A.B., M.D., 126 East Thirty-seventh Street, New York City. Professor of Clinical Surgery, Cornell Medical School; Associate Surgeon, New York Hospital.

1918. HODGE, EDWARD BLANCHARD, A.B., M.D., 2019 Spruce Street, Philadelphia, Pa. Surgeon to Presbyterian and Children's Hospitals; Associate Surgeon to Pennsylvania Hospital; Associate Surgeon to Widener Memorial School.

1923. HOMANS, JOHN, A.M., M.D., Peter Bent Brigham Hospital, 721 Huntington Avenue, Boston, Mass. Instructor in Surgery, Harvard Medical School; Surgeon to the Peter Bent Brigham Hospital.

ELECTED

1924. HORSLEY, J. SHELTON, M.D., St. Elizabeth's Hospital, Richmond, Va. Attending Surgeon, St. Elizabeth's Hospital.

1918. HUBBARD, JOSHUA C., M.D., 86 Bay State Road, Boston, Mass. Chief of Third Surgical Service, Boston City Hospital; Surgeon, Newton Hospital; Consulting Surgeon, Norwood and Leonard Morse Hospitals.

†1901. HUNTINGTON, THOMAS W., A.B., M.D., LL.D., 960 Mills Building, San Francisco, California. Professor of Clinical and Operative Surgery (Emeritus), College of Medicine, University of California; Consulting Surgeon, St. Luke's Hospital, San Francisco; Chief Surgeon, Western Pacific R. R. *Vice-President*, 1906; *President*, 1917; *Member of Council*, 1918–20.

1919. HUPP, FRANK LE MOYNE, A.M., M.D., 61 Fourteenth Street, Wheeling, W. Va. Attending Surgeon, Ohio Valley General Hospital; Consulting Surgeon, Reynolds Memorial Hospital, Glendale.

†1909. HUTCHINSON, JAMES P., M.D., Media, Pa.

†1913. HUTCHISON, JAMES ALEXANDER, C.B.E., M.D., (McGill), L.R.C.P. and S. (Edin.), 354 MacKay Street, Montreal. Emeritus Professor of Surgery and Clinical Surgery, McGill University; Consulting Surgeon, Montreal General and Royal Victoria Hospitals.

†1913. JACKSON, JABEZ N., A.M., M.D., 425 Argyle Building, Kansas City, Mo. Surgeon, Christian Church and Kansas City General Hospitals.

†1905. JOHNSON, ROBERT W., A.B., M.D., 101 W. Franklin Street, Baltimore, Md. Professor of Principles and Practice of Surgery, Baltimore Medical College; Surgeon to Maryland General Hospital, Church Home and Infirmary, and St. Agnes' Hospital. Retired.

†1901. JONAS, A. F., M.D., 612 Omaha Loan Building, Omaha, Neb. Head of the Department in Surgery, College of Medicine, University of Nebraska (formerly Omaha Medical College); Surgeon-in-Chief, Omaha Methodist Episcopal Hospital; Captain, M.C. *Vice-President*, 1906.

1917. JONES, DANIEL FISKE, A.B., M.D., 195 Beacon Street, Boston. Mass. Associate in Surgery, Courses for Graduates, H.M.S.; Visiting Surgeon to Massachusetts General Hospital, and M.E. Deaconess' Hospital; Consulting Surgeon to the New England Hospital for Women and Children, Brockton City Hospital, Union Hospital of Fall River, Sturdy

ELECTED

Hospital of Attleboro and the Addison Gilbert Hospital of Gloucester, Mass.

1910. JOPSON, JOHN HOWARD, M.D., *Recorder*, 1824 Pine Street, Philadelphia, Pa. Professor of Clinical Surgery, University of Pennsylvania; Professor of Surgery, the Graduate School of Medicine, University of Pennsylvania; Surgeon to Presbyterian Hospital and Children's Hospital; Consulting Surgeon to Norristown State Hospital, Shriner's Hospital for Crippled Children and Philadelphia Home for Incurables.

1914. JUDD, EDWARD STARR, M.D., Rochester, Minn. Surgeon to Mayo Clinic. *Vice-President*, 1925.

†1899. KAMMERER, FREDERIC, M.D., Lohn, Kehrsatz Ct., Berne, Switzerland. Professor of Clinical Surgery, Columbia University; Consulting Surgeon to the Lenox Hill Hospital, and to St. Francis Hospital, New York.

1913. KANAVEL, ALLEN B., A.B., M.D., 54 East Erie Street, Chicago, Ill. Professor of Surgery, Northwestern University Medical School; Attending Surgeon to Wesley Memorial and Cook County Hospitals.

*†1880. KEEN, WILLIAM WILLIAMS, A.M., M.D., LL.D., Hon. F.R.C.S. (Eng., Edin. and Ireland), Hon. Ph.D. (Upsala), Hon. Sc.D. (Jeff. and Harv.), Hon. F.A.C.S., The Touraine, 1520 Spruce Street, Philadelphia. Emeritus Professor of Surgery in the Jefferson Medical College; Consulting Surgeon to the Jefferson Medical College Hospital and to the Woman's and St. Agnes' Hospitals; Membre Correspondant Etranger de la Société de Chirurgie de Paris and of the Académie de Médecine de Paris; Membre Honoraire de la Société Belge de Chirurgie; Honorary Member, Clinical Society of London; Royal Society of Medicine, London; Doctor honoris causa of the University of Paris; Bigelow Medal of the Boston Surgical Society, etc. *President*, 1898; *Vice-President*, 1892; *Member of Council*, 1899–1901.

1923. KELLER, WILLIAM L., M.D., Walter Reed General Hospital, Washington, D. C. Colonel, M.C., U. S. A.; Professor of Military Surgery, Army Medical School, Army Medical Center, Washington, D. C.; Chief of the Surgical Service, Walter Reed General Hospital.

1924. KERR, HARRY HYLAND, M.D., C.M. (McGill), 1744 N. Street, N.W., Washington, D.C. Professor of Neurosurgery and Clinical Professor of Surgery, George Washington University; Attending Surgeon, Garfield Memorial and Children's

ELECTED

Hospitals; Consulting Surgeon, Providence and Columbia Hospitals; formerly Attending Surgeon, Freedman's Hospital, Washington, D. C.; Lieut.-Colonel, M.R.C.

1925. LAHEY, FRANK H., M.D., 118 Bay State Road, Boston, Mass. Surgeon, N. E. Deaconess' Hospital and N. E. Baptist Hospital.

1916. LAW, ARTHUR AYER, M.D., F.A.C.S., *Vice-President*, 420 Syndicate Building, Minneapolis, Minn. Associate Professor of Surgery, University of Minnesota Medical School, and Mayo Foundation; Surgeon to the Northwestern Hospital; Surgeon to Glen-Lake Sanatorium; Surgeon to and Assistant Chief of the University Hospital; Lieut.-Col., M.C. (discharged), Commanding University of Minnesota Base Hospital No. 26 in A. E. F.

1919. LEE, BURTON JAMES, PH.B., M.D., F.R.C.S., 128 East Seventy-third Street, New York. Clinical Professor of Surgery, Cornell Medical School; Associate Attending Surgeon, New York Hospital; Consulting Surgeon, Sharon (Connecticut) Hospital; Attending Surgeon, Memorial Hospital.

1923. LEE, WALTER ESTELL, M.D., F.A.C.S., 905 Pine Street, Philadelphia, Pa. Associate Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon to the Pennsylvania, Germantown, Bryn Mawr and Children's Hospitals and the Associated Medical Clinics.

1912. LEWIS, DEAN D., M.D., Johns Hopkins Hospital, Baltimore, Md. Professor of Surgery, Johns Hopkins University.

1910. LILIENTHAL, HOWARD, A.B., M.D., 52 East Eighty-second Street, New York. Consulting Surgeon, Mount Sinai Hospital, Bellevue Hospital, Hospital for Joint Diseases, and New York Infirmary for Women and Children; Professor of Clinical Surgery, Cornell Medical College; formerly Lieut.-Col., M.C., U. S. A.

†1908. LOTHROP, HOWARD AUGUSTUS, A.M., M.D., 101 Beacon Street, Boston, Mass. Assistant Professor of Surgery, Harvard Medical School; Surgeon-in-Chief, Boston City Hospital.

1916. LOWER, WILLIAM E., M.D., Euclid Avenue and Ninety-third Street, Cleveland, Ohio. Associate Professor of Genito-urinary Surgery, School of Medicine, Western Reserve University; Surgeon, Cleveland Clinic Hospital; Associate Surgeon in charge of Genito-urinary Surgery, Lakeside Hospital; Visiting Surgeon, Lutheran Hospital.

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1910. LUND, FRED BATES, M.D., 527 Beacon Street, Boston, Mass. Surgeon-in-Chief, Boston City Hospital; Consulting Surgeon, Burbank Hospital, Fitchburg, Newton Hospital, Newton, Choate Memorial Hospital, Woburn, Mass; lately Lieut.-Col., M.C., U. S. A.

1917. LYLE, HENRY H. M., M.D., 33 East Sixty-eighth Street, New York City. Professor of Clinical Surgery, College of Physicians and Surgeons, Columbia University; Attending Surgeon, St. Luke's Hospital.

†1901. MCARTHUR, L. L., M.D., F.A.C.S., 122 S. Michigan Boulevard, Chicago, Ill. Assistant Professor of Clinical Surgery, Rush Medical College; Senior Surgeon to St. Luke's and Michael Reese Hospitals. *President*, 1922. *Member of Council*, 1923–25.

1923. MCGUIRE, EDGAR R., M.D., F.A.C.S., 622 Delaware Avenue, Buffalo, N. Y. Professor of Surgery, University of Buffalo; Surgeon, Buffalo General Hospital; Surgeon-in-Chief, Buffalo City Hospital.

1917. MCGUIRE, STUART, M.D., LL.D., 1000 West Grace Street, Richmond, Va. President of Medical College of Virginia; Surgeon to St. Luke's Hospital; Consulting Surgeon to Memorial Hospital.

1925. MCKENNA, HUGH, B.S., M.D., 920 Monroe Building, Chicago, Ill. Assistant Clinical Professor of Surgery, Rush Medical College; President of the Staff and Attending Surgeon, St. Joseph's Hospital; Attending Surgeon, Cook County Hospital.

‡1918. MCWILLIAMS, CLARENCE A., M.A., M.D., 19 East Sixty-fifth Street, New York, N. Y. Associate Surgeon, Fifth Avenue and New York Polyclinic Hospitals; Attending Surgeon, New York Skin and Cancer Hospital.

†1914. MACLAREN, MURRAY, B.A., M.B.C.M., M.D., M.R.C.S. (Eng.), C.M.G., C.O.A. (Commander, Order of Avis), LL.D., M.P. (Member of Parliament), 75 Coburg Street, St. John's, N.B., Canada. Surgeon to the General Public Hospital; Colonel, C.A.M.C.

1920. MAES, URBAN, M.D., 1671 Octavia Street, New Orleans, Louisiana. Professor of Clinical and Operative Surgery, Tulane University of Louisiana School of Medicine; Senior Visiting Surgeon, Charity Hospital; Senior Associate in Surgery, Touro Infirmary.

ELECTED

†1898. MARTIN, EDWARD, A.M., M.D., LL.D., D.Sc., Media, Pa. Professor of Surgical Physiology, University of Pennsylvania; Consulting Surgeon to State Hospitals, Norristown and Wernersville, Pa.; Colonel, M.R.C., U. S. A.

1915. MARTIN, WALTON, PhB., M. D., 230 East Forty-ninth Street, New York. Professor of Clinical Surgery, Columbia University; Attending Surgeon, St. Luke's Hospital; Consulting Surgeon, White Plains Hospital and Loomis Sanatorium.

†1895. MASTIN, WILLIAM MCDOWELL, M.D., LL.D., Northeast corner of Joachim and Conti Streets, Mobile, Ala. Surgeon to the Mobile City Hospital and Providence Infirmary.

†1895. MATAS, RUDOLPH, M.D., LL.D., 2255 St. Charles Avenue, New Orleans, Louisiana. Professor of Surgery, Tulane University of Louisiana, School of Medicine; Professor (Emeritus), Graduate Medical School, Tulane University; Chief, Surgical Division, Touro Infirmary; Senior Visiting Surgeon, Charity Hospital; Consulting Surgeon, Eye, Ear, Nose and Throat Hospital; Major, Reserve Corps, U. S. Army. *President*, 1909; *Vice-President*, 1901.

1918. MATHEWS, FRANCIS STUART, B.S., M.D., F.A.C.S., 49 West Fifty-second Street, New York. Professor of Clinical Surgery, Columbia University; Surgeon to St. Luke's Hospital and St. Mary's Hospital for Children; Consulting Surgeon to the Home for Incurables.

1903. MAYO, CHARLES H., A.M., M.D., LL.D., D.Sc., F.A.C.S., Rochester, Minn. Professor of Surgery, Medical School, University of Minnesota; Surgeon to Mayo Clinic (St. Mary's Hospital).

1899. MAYO, WILLIAM J., A.M., M.D., M.D. in Chir., Trinity College, Dublin, D.Sc. (Columbia, Mich., Leeds, Harvard), LL.D. (Toronto, McGill, Pennsylvania, Maryland, Pittsburgh), F.R.C.S. (Eng., Edinburgh, Ireland), F.A.C.S., Sigma Xi, Phi Beta Kappa, 701 Fourth Street, S. W., Rochester, Minnesota. Surgeon and Chief of Staff to Mayo Clinic. *Vice-President*, 1903; *President*, 1913.

†1901. MEYER, WILLY, M.D., 700 Madison Avenue, New York. Emeritus Professor of Surgery in the New York Post-Graduate Medical School; Consulting Surgeon to the Lenox Hill Hospital, Post-Graduate Hospital, the New York Infirmary for Women and Children, New York Skin and Cancer Hospital, Hospital for Deformities and Joint Diseases, Montefiore and Glens Falls Hospital.

ELECTED

1919. Miller, Robert Talbott, A.B., M.D., 9 East Chase Street, Baltimore, Md. Associate Professor of Surgery, Johns Hopkins University; Associate Surgeon, Johns Hopkins Hospital.

1917. Mitchell, Charles F., M.D., 2003 Pine Street, Philadelphia, Pa. Surgeon-in-Chief, Pennsylvania Hospital; Attending Surgeon to the Germantown and the Bryn Mawr Hospitals; Consulting Surgeon, Chestnut Hill Hospital.

1912. Mitchell, James F., A.B., M.D., 1344 Nineteenth Street, Washington, D. C. Clinical Professor of Surgery, Georgetown and George Washington Universities; Chief Surgeon, Emergency Hospital. *Vice-President*, 1920.

1924. Mixter, Charles G., S.B., M.D., 270 Commonwealth Avenue, Boston. Assistant Surgeon, Children's Hospital, Boston.

1920. Mixter, William Jason, S.B., M.D., F.A.C.S., 270 Commonwealth Avenue, Boston, Massachusetts. Assistant Visiting Surgeon, Massachusetts General Hospital; Assistant in Surgery, Graduate School of Medicine, Harvard University; Surgeon to the Staff, Anna Jaques Hospital, Newburyport; Consulting Surgeon, Leonard Morse Hospital, Natick, Charitable Eye and Ear Infirmary, Boston, Cable Memorial Hospital, Ipswich; Lieut.-Col., Med. O.R.C.

†1896. Monks, George Howard, A.B. (Harvard), M.D. (Harvard), M.R.C.S. (Eng.), 51 Commonwealth Avenue, Boston. Consulting Surgeon, Boston City Hospital; Professor of Oral Surgery, Harvard Dental School. *Vice-President*, 1909.

1925. Moore, Edward Clarence, M.D., Moore-White Clinic, 511 South Bonnie Brae Street, Los Angeles, Calif. Consulting Surgeon, Children's Hospital and Los Angeles General Hospital; Surgeon to Hollywood Hospital, Hospital of the Good Samaritan, California Lutheran Hospital; Vice-Chairman, California Lutheran Hospital.

1914. Moschcowitz, Alexis V., M.D., 925 Madison Avenue, New York, N. Y. Professor of Clinical Surgery, College of Physicians and Surgeons, Columbia University; Attending Surgeon, Mount Sinai Hospital; Consulting Surgeon, Israel Zion Hospital.

†1904. Mudd, Harvey, G., M.D., Humboldt Building, St. Louis, Mo. Emeritus Clinical Professor of Surgery, Medical Department, Washington University; Surgeon to St. Luke's Hospital; Consulting Surgeon to the Barnard Free Skin and Cancer Hospital. *Vice-President*, 1920.

ELECTED

1919. MÜLLER, GEORGE P., M.D., 1930 Spruce Street, Philadelphia, Pa. Professor of Clinical Surgery, University of Pennsylvania; Surgeon to the University and Misericordia Hospitals.

†1913. MURPHY, FRED T., A.B., M.A., M.D., 2248 Penobscot Building, Detroit, Michigan.

†1898. MURRAY, FRANCIS W., M.D., 7 West Forty-third Street, New York City. Consulting Surgeon, New York Hospital, St. Luke's Hospital, Paul Kimball Hospital, Lakewood, and Goshen Emergency Hospital.

†1903. NEILSON, THOMAS R., M.D., 1937 Chestnut Street, Philadelphia, Pa. Surgeon Emeritus to the Episcopal Hospital; Consulting Surgeon to St. Christopher's Hospital for Children; Emeritus Professor of Genito-urinary Surgery in the University of Pennsylvania.

†1900. OLIVER, J. C., M.D., 628 Elm Street, Cincinnati, Ohio. Emeritus Professor of Surgery, Medical College, University of Cincinnati; Surgeon to the Christ Hospital.

1915. OLMSTED, INGERSOLL, M.B., 215 James Street, South, Hamilton, Ontario. Surgeon to City Hospital.

1923. PALMER, DUDLEY WHITE, B.S., M.D., F.A.C.S., 707 Race Street, Pearl Market Bank Building, Cincinnati, Ohio. Associate Professor of Clinical Surgery, University of Cincinnati, Medical Department; Attending Surgeon, Cincinnati General Hospital and Christ Hospital.

†1899. PARHAM, FREDERICK WILLIAM, M.D., 3513 Prytania Street, New Orleans, La. Professor of General Clinical Surgery, New Orleans Polyclinic (Post-Graduate Department of Tulane University of Louisiana); Chief of Staff to Charity Hospital and to Touro Infirmary.

†1893. PARMENTER, JOHN, M.D., Ridgecroft, Geneva, N. Y. Late Professor of Clinical Surgery, Medical Department, University of Buffalo.

1908. PECK, CHARLES HOWARD, M.D., 107 East Sixty-seventh Street, New York City. Professor of Clinical Surgery, Columbia University; Senior Attending Surgeon to the Roosevelt Hospital; Consulting Surgeon to French Hospital, Memorial Hospital and Hospital for Ruptured and Crippled, New York City; Vassar Brothers Hospital, Poughkeepsie, N. Y.; White Plains Hospital, White Plains, N. Y.; Stam-

ELECTED

ford Hospital, Stamford, Conn.; Greenwich Hospital, Greenwich, Conn. *Treasurer*, 1915-1924.

1919. PERCY, NELSON MORTIMER, M.D., F.A.C.S., 2106 Sedgwick Street, Chicago, Ill. Professor of Clinical Surgery, Medical Department of the University of Illinois; Attending Surgeon to the Augustana and St. Mary's of Nazareth Hospitals.

1925. PFEIFFER, DAMON B., A.B., M.D., 1822 Pine Street, Philadelphia, Pa. Assistant Professor of Surgery, Graduate School, University of Pennsylvania; Surgeon to Methodist and Abington Memorial Hospitals; Associate Surgeon, Presbyterian Hospital; Assistant Surgeon, Lankenau Hospital.

1917. PHEMISTER, DALLAS B., B.S., M.D., 122 South Michigan Avenue, Chicago, Ill. Associate Professor in Surgery, Rush Medical College; Associate Attending Surgeon, Presbyterian Hospital.

†1889. PILCHER, LEWIS STEPHEN, A.M., M.D., LL.D. (Dickinson and University of Michigan), 145 Gates Avenue, Brooklyn, N. Y. Consulting Surgeon to the St. John, Norwegian, Jewish, Bushwick and Deaconess Hospitals. *Vice-President*, 1893, 1914; *President*, 1918; *Member of Council*, 1919-21.

1914. PLUMMER, SAMUEL C., A.B., M.D., 4539 Oakenwald Avenue, Chicago, Ill. Attending Surgeon, St. Luke's Hospital; Chief Surgeon, C. R. I. & P. Railway. Co.

1914. POOL, EUGENE HILLHOUSE, A.B., M.D., *Treasurer*, 107 East Sixtieth Street, New York, N. Y. Clinical Professor of Surgery, Columbia University; Attending Surgeon, New York Hospital; Consultant Surgeon to French Hospital, New York Orthopedic, Manhattan State Hospital, Home for Incurables and United Hospital at Portchester, New York Eye and Ear, and Hospital for Ruptured and Crippled.

1904. PORTER, CHARLES ALLEN, A.M., M.D., 116 Beacon Street, Boston, Mass. Chief of the West Surgical Service of the Massachusetts General Hospital; Homans Professor of Surgery, Harvard Medical School.

†1915. PORTER, MILES FULLER, M.A., M.D., 2326 Fairfield Avenue, Wayne, Ind. Professor of Surgery in the Indiana University School of Medicine; Surgeon to St. Joseph's Hospital; Consulting Surgeon in the Indiana School for Feeble-minded Youths.

1908. PRIMROSE, ALEXANDER, C.B., M.B., C.M. (Edin.), M.R.C.S. (Eng.), *Vice-President*, 100 College Street, Toronto, Canada. Consulting Surgeon to the Toronto General Hospital and

ELECTED

Consulting Surgeon to the Hospital for Sick Children, Toronto; Dean of the Faculty of Medicine and Professor of Clinical Surgery in the University of Toronto. *Vice-President*, 1922.

1926. Reid, Mont Rogers, A.B., M.D., Walsh Place and Edwards Road, Hyde Park, Cincinnati, Ohio. Associate Professor of Surgery, University of Cincinnati College of Medicine; Assistant Director, Surgical Service, Cincinnati General Hospital and Children's Hospital.

1923. Richardson, Edward P., A.B., M.D., Massachusetts General Hospital, Boston, Mass. Assistant Professor of Surgery, Harvard Medical School; Chief of Third Surgical Service, Massachusetts General Hospital.

1924. Ritchie, Harry P., Ph.B., M.D., 914 Lowry Building, St. Paul, Minn. Associate Professor of Surgery, University of Minnesota Medical School; Attending Surgeon, University Hospital, Minneapolis; Chief, Department of Surgery, Wilder Dispensary, St. Paul; Staff of St. Luke's and Miller Hospitals, St. Paul.

1901. Rixford, Emmet, B.S., M.D., 1795 California Street, San Francisco, Calif. Professor of Surgery in Stanford University School of Medicine; Surgeon to the San Francisco and Lane Hospitals; Member of the Society of Clinical Surgery. *Vice-President*, 1905.

1924. Rodman, J. Stewart, M.D., 1309 Medical Arts Building, Philadelphia. Professor of Surgery, Woman's Medical College of Pennsylvania; Surgeon-in-Chief, Woman's Medical College Hospital; Attending Surgeon, Bryn Mawr Hospital; Associate Surgeon, Presbyterian Hospital; Major, M.R.C.

1917. Rowan, Charles J., A.B., M.D., 227 North Clinton Street, Iowa City, Iowa. Professor and Head of Department of Surgery, College of Medicine, State University of Iowa; Director of Surgery, University Hospital.

1922. Royster, Hubert A., A.B., M.D., 423 Fayetteville Street, Raleigh, N. C. Surgeon to Rex Hospital; Surgeon-in-Chief to St. Agnes Hospital; Consulting Surgeon to Dix Hill State Hospital for the Insane.

†1882. Rushmore, John Dikeman, M.D., 129 Montague Street, Brooklyn, N. Y. Professor of Operative and Clinical Surgery, Emeritus, Long Island College Hospital; Surgeon to St. Peter's Hospital; Consulting Surgeon to Long Island College Hospital, Kings County Hospital, Swedish and Lutheran Hospitals.

ELECTED

†1909. SCUDDER, CHARLES L., M.D., A.B., PH.B., 144 Commonwealth Avenue., Boston, Mass. Consulting Surgeon, Massachusetts General Hospital; former Assistant Professor of Surgery, Harvard University Medical School.

1919. SEELIG, M. G., A.B., M.D., 4952 Park View Place, St. Louis, Mo. Professor of Clinical Surgery, Washington University School of Medicine; Assistant Surgeon, Barnes Hospital; Visiting Surgeon to the Jewish Hospital.

†1902. SHEPHERD, FRANCIS J., M.D., LL.D., F.R.C.S. (Eng.) F.R.C.S. (Edin.) (Hon.), F.A.C.S. (Hon.), 152 Mansfield Street, Montreal, Canada. Emeritus Professor in McGill University; Consulting Surgeon to the Montreal General and Royal Victoria Hospitals, Montreal.

1923. SHIPLEY, ARTHUR M., M.D., SC.D., 1827 Eutaw Place, Baltimore, Md. Professor of Surgery and Head of the Department of Surgery, University of Maryland; Chief Surgeon, University Hospital, City Hospitals and Sydenham Hospital; Visiting Surgeon, Union Memorial Hospital, Hospital for Women of Maryland, Mercy Hospital and Maryland General Hospital.

1926. SIMMONS, CHANNING C., M.D., 205 Beacon Street, Boston, Mass. Instructor in Surgery, Harvard Medical School; Visiting Surgeon, Massachusetts General Hospital; Surgeon, Collis P. Huntington Memorial Hospital.

1920. SISTRUNK, WALTER E., M.D., F.A.C.S., Rochester, Minn., Surgeon to Saint Mary's, Kahler and Colonial Hospitals (Mayo Clinic), Associate Professor of Surgery, Mayo Foundation, University of Minnesota.

1918. SMITH, REA, M.D., The Medical Office Building, 1136 West Sixth Street, Los Angeles, Calif. Surgeon, Hospital of the Good Samaritan, Methodist Hospital; Los Angeles General Hospital, California Lutheran Hospital.

1919. SPEED, KELLOGG, S.B., M.D., F.A.C.S., 122 South Michigan Avenue, Chicago, Ill. Assistant Professor of Clinical Surgery, Rush Medical College of the University of Chicago; Associate Attending Surgeon, Presbyterian Hospital; Attending Surgeon, Cook County, Provident and Highland Park Hospitals.

1922. SPEESE, JOHN, M.D., 1832 Spruce Street, Philadelphia, Pa. Associate Professor of Surgery, Graduate School, University of Pennsylvania; Surgeon to the Presbyterian Hospital, Children's Hospital, and the Philadelphia Home for Incurables.

ELECTED

1918. STARR, CLARENCE LESLIE, M.B., M.D., LL.D., 182 Bloor Street, West, Toronto, Canada. Professor of Surgery, University of Toronto; Surgeon-in-Chief, Toronto General Hospital; Consultant in Surgery, Hospital for Sick Children; Surgeon to St. John's Hospital; Lieut.-Col.-Consultant, C.A.M.C.

1914. STARR, FREDERIC NEWTON GISBORNE, C.B.E., M.B., M.D., C.M., 112 College Street, Toronto, Canada. Professor of Clinical Surgery, University of Toronto; Consulting Surgeon to Hospital for Sick Children, and Surgeon to St. John's Hospital for Women; Senior Surgeon, Toronto General Hospital.

1915. STEWART, GEORGE DAVID, M.D., 61 West Fiftieth Street, New York. President, New York Academy of Medicine; Professor of Surgery, New York University Medical School; Visiting Surgeon to St. Vincent's and Bellevue Hospitals; Consulting Surgeon to Hospital for Ruptured and Crippled of New York; to St. Joseph's Hospital, Yonkers; to St. Mary's Hospital, East Orange, N. J.; to South Side Hospital, Babylon, L. I.; to Englewood Hospital, Englewood, N. J., and to the United Hospital, Portchester, N. Y.

†1909. STILLMAN, STANLEY, M.D., Stanford University Hospital, San Francisco, Calif. Professor of Surgery, Stanford University Medical School; Commander, M.C., U.S.N.R.F.; Surgeon to Lane and Stanford University Hospitals. *Vice-President*, 1924.

1926. STONE, HARVEY BRINTON, M.D., 18 West Franklin Street, Baltimore, Md. Associate in Clinical Surgery, Johns Hopkins Medical School; Assistant Visiting Surgeon, Johns Hopkins Hospital; Visiting Surgeon, Church Home and Infirmary, Union Memorial Hospital, Hospital for the Women of Maryland, Bon Secours Hospital and Mt. Sinai Hospital.

1923. ST. JOHN, FORDYCE BARKER, B.S., M.D., Presbyterian Hospital, 41 East Seventieth Street, New York City. Associate Professor of Surgery, College of Physicians and Surgeons; Visiting Surgeon to the Presbyterian Hospital.

†1909. SUMMERS, JOHN EDWARD, M.D., Bankers Reserve Life Building, Nineteenth and Douglas Streets, Omaha, Neb. Professor of Clinical Surgery, University of Nebraska Medical School; Surgeon to the University Hospital and to the Clarkson Memorial and the Douglas County Hospitals, Omaha, Neb.

ELECTED

1920. TAYLOR, ALFRED S., B.P., A.M., M.D., 115 W. Fifty-fifth Street, New York. Consulting Surgeon, Fordham Hospital; Consulting Neurological Surgeon to St. Luke's Hospital, N. Y.; Neurological Surgeon, Hospital for Ruptured and Crippled; Consulting Surgeon to Memorial Hospital, New York City, Tarrytown Hospital, New York, White Plains Hospital, New York and Overlook Hospital, Summit, N. J.

†1900. TAYLOR, WILLIAM J., M.D., 1825 Pine Street, Philadelphia. Surgeon to the Orthopædic Hospital and Infirmary for Nervous Diseases; Consulting Surgeon to St. Agnes' Hospital, to the West Philadelphia Hospital for Women and to the Woman's Hospital; Member of the International Surgical Society.

1916. TERRY, WALLACE I., B.S., M.D., 2721 Broadway, San Francisco, Calif. Professor of Surgery, University of California; Chief Surgeon, University of California Hospital, San Francisco.

1909. THOMPSON, JAMES E., M.B., B.S. (London), F.R.C.S. (Eng.) F.A.C.S., 3224 Broadway, Galveston, Texas. Professor of Surgery in the University of Texas; Surgeon to the John Sealy Hospital. *Vice-President*, 1922.

1920. TINKER, MARTIN B., B.S., M.D., Savings Bank Building, Ithaca, N. Y. Surgeon to the Ithaca City Hospital and the Cornell Infirmary.

1925. TROUT, HUGH HENRY, M.D., F.A.C.S., Jefferson Hospital, Roanoke, Va. Surgeon-in-Chief, Jefferson Hospital.

1918. TRUESDALE, PHILEMON EDWARDS, M.D., 151 Rock Street, Fall River, Mass. Surgeon to St. Anne's and Highland Hospitals; Consulting Surgeon to Fall River City Hospital.

†1882. VANDER VEER, ALBERT, A.M., M.D., PH.D., LL.D., 28 Eagle Street, Albany, N. Y. Senior Consulting Surgeon, Albany Hospital; Consulting Surgeon, Albany Free Dispensary Association, Benedictine Hospital, Kingston, N. Y., and Champlain Valley Hospital, Plattsburgh, N. Y. *President*, 1905; *Vice-President*, 1898; *Member of Council*, 1907.

1910. VANDER VEER, EDGAR ALBERT, Ph.B., M.D., 28 Eagle Street, Albany, N. Y. Attending Surgeon, Albany Hospital; Consulting Surgeon, Champlain Valley Hospital and Benedictine Hospital, Kingston, N. Y.

†1902. VAUGHAN, GEORGE TULLY, M.D., LL.D., 1718 I Street, Washington, D. C. Professor of Principles and Practice of Surgery and Head of the Surgical Department, George-

ELECTED

town University; Chief Surgeon, Georgetown University Hospital; Surgeon, Tuberculosis Hospital; Consulting Surgeon, Government Hospital for the Insane, Washington, D. C., U. S. Public Health Service and Washington Asylum Hospital; Commander, M.C., Reserve Force, U. S. N.

1921. VAUGHAN, J. WALTER, A.B., M.D., 987 East Jefferson Avenue, Detroit, Mich. Associate Attending Surgeon to Harper Hospital; Surgeon, Detroit Diagnostic Hospital.

1920. VERDI, WILLIAM FRANCIS, A.M., M.D., 27 Elm Street, New Haven, Connecticut. Clinical Professor of Surgery, Yale University School of Medicine; Chief Surgeon, Hospital of St. Raphael; Attending Surgeon, New Haven Hospital; Consulting Surgeon, St. Vincent's Hospital, Bridgeport; St. Mary's Hospital, Waterbury; Middlesex Hospital, Middletown.

1922. VINCENT, BETH, A.B., M.D., 295 Beacon Street, Boston, Mass. Assistant Visiting Surgeon, Massachusetts General Hospital; Consulting Surgeon, Infants Hospital, Boston.

1918. WAINWRIGHT, JONATHAN M., A.B., A.M., M.D., 912 Clay Avenue, Scranton, Pa. Surgeon-in-Chief, Moses Taylor Hospitals (Scranton and Buffalo); Chief Surgeon, D. L. & W. R. R. Co. and Glen Alden Coal Co.

1909. WALKER, JOHN B., M.D., 51 East Fiftieth Street, New York City. Consulting Surgeon to Bellevue Hospital and Hospital for Ruptured and Crippled, New York; Professor of Clinical Surgery, College of Physicians and Surgeons, Columbia University, N. Y.

†1882. WARREN, JOHN COLLINS, M.D., LL.D. (Jefferson, Harvard and McGill), Hon. F.R.C.S. (Eng. and Edin.), 58 Beacon Street, Boston, Mass. Moseley Professor of Surgery, Emeritus, Harvard University; Consulting Surgeon to the Massachusetts General Hospital; Member, Harvard Cancer Commission. *President*, 1896; *Vice-President*, 1890; *Member of Council*, 1897.

†1896. WATSON, FRANCIS SEDGWICK, A.B., M.D., South Dartmouth, Mass. Surgeon to the Boston City Hospital; Instructor in Genito-urinary Surgery, Harvard Medical School.

1913. WATTS, STEPHEN H., M.A., M.D., LL.D., Department of Surgery, University of Virginia, Charlottesville, Va. Professor of Surgery and Gynecology in the University of Virginia; Surgeon-in-Chief, University of Virginia Hospital.

ELECTED

†1889. WEIR, ROBERT F., A.M., M.D., Hon. F.R.C.S. (Eng.), Cor. Mem. Surg. Soc., Paris, 16 East Ninety-sixth Street, New York. Professor of Surgery in the College of Physicians and Surgeons, Columbia University; Surgeon to the Roosevelt Hospital. *President*, 1899; *Member of Council*, 1893–1896, 1901–1903.

1921. WHIPPLE, ALLEN O., B.S., M.D., Presbyterian Hospital, 41 East Seventieth Street, New York, N. Y. Professor of Surgery, Columbia University; Director of Surgical Service, Presbyterian Hospital.

1926. WHITTEMORE, WYMAN, S.B., M.D., 199 Beacon Street, Boston, Mass. Instructor in Surgery, Harvard Medical School; Associate Surgeon, Massachusetts General Hospital; Visiting Surgeon, Beth Israel Hospital; Consulting Surgeon, Massachusetts Eye and Ear Infirmary, Malden, Sturdy Memorial and Peterboro Hospitals.

1921. WILLIAMS, HUGH, A.B., M.D., 301 Beacon Street, Boston, Mass. Assistant, Harvard Graduate School of Medicine; Visiting Surgeon, Massachusetts General Hospital.

†1914. WINSLOW, RANDOLPH, A.B., M.D., A.M., LL.D., St. Johns College and University of Maryland, F.A.C.S., 1900 Mt. Royal Terrace, Baltimore, Md. Emeritus Professor of Surgery, University of Maryland.

1918. WOOD, ALFRED CONARD, M.D., 2035 Walnut Street, Philadelphia, Pa. Assistant Professor of Surgery, University of Pennsylvania; Surgeon to the University, Philadelphia General, Howard and Rush Hospitals.

†1901. WOOLSEY, GEORGE, M.D., 117 East Thirty-sixth Street, New York City. Professor of Clinical Surgery, Cornell University Medical College; Consulting Surgeon to Bellevue Hospital, Memorial Hospital, Peekskill Hospital, Peekskill, N. Y., and St. John's Riverside Hospital, Yonkers, N. Y.

1916. YATES, J. L., PH.B., B.S., M.D., F.A.C.S., 141 Wisconsin Street, Milwaukee, Wis. Attending Surgeon, Milwaukee County Hospital, and Columbia Hospital.

SENIOR FELLOWS OF THE ASSOCIATION

Abbe, Robert, M.D., 40 West Fifty-ninth Street, New York.

Armstrong, George E., M.D., C.M., C.M.G., LL.D., D.Sc., 320 Mountain Street, Montreal.

Barrow, David, M.D., 190 N. Upper, Lexington, Ky.

‡Barton, James M., A.M., M.D., 32 North New Hampshire Avenue, Atlantic City, N. J.

Binnie, John F., M.A., M.B., C.M., 917 Rialto Building, Kansas City, Mo.

Blake, John Babst, A.M., M.D., 311 Beacon Street, Boston, Mass.

Blake, Joseph A., A.B., PhB., M.A. (Hon.), M.D., F.A.C.S., 116 East Fifty-third Street, New York City.

Brewer, George E., M.D., LL.D., F.A.C.S., F.R.C.S. (Ireland), 19 East Sixty-fifth Street, New York City.

Brinsmade, W. B., M.D., 166 Columbus Heights, Brooklyn, N. Y.

Bunts, Frank E., M.D., Clinic Building, Cleveland, Ohio.

Carmalt, William H., A.M., M.D., 261 St. Ronan Street, New Haven, Conn.

Carson, Norman Bruce, M.D., 7006 Maryland Drive, St. Louis, Mo.

Codman, Ernest Amory, 227 Beacon Street, Boston, Mass.

Coley, William B., M.D., The Medical Chambers, 114 East Fifty-fourth Street, New York City.

Crile, George W., M.D., Euclid Avenue and Ninety-third Street, Cleveland, Ohio.

Cushing, Hayward Warren, A.B., M.D., 70 Commonwealth Avenue, Boston, Mass.

Da Costa, J. Chalmers, M.D., 2045 Walnut Street, Philadelphia, Pa.

Deaver, Harry C., M.D., 337 South Eighteenth Street, Philadelphia, Pa.

Dennis, Frederic Shepard, M.D., F.R.C.S. (Eng.), 1136 Fifth Avenue, New York, N. Y.

Dowd, Charles N., A.M., M.D., Sc.D., 138 West Fifty-eighth Street, New York, N.Y.

Estes, William Lawrence, A.M., M.D., Bethlehem, Pa.

Eve, Duncan, A.M., M.D., Eve Building, Nashville, Tenn.

Finney, John M. T., M.D., 1300 Eutaw Place, Baltimore, Md.

Freeman, Leonard, B.S., M.A., M.D., 2611 East Seventh Avenue, Denver, Col.

Gage, Homer, A.M., M.D., 72 Pearl Street, Worcester, Mass.

Gibson, Charles L., M.D., 72 East Fifty-fourth Street, New York, N. Y.

Gay, George W., A.M., M.D., Chestnut Hill, Boston, Mass.

Huntington, Thomas W., A.B., M.D., LL.D., 960 Mills Building, San Francisco, Calif.

Hutchison, James A., 354 MacKay Street, Montreal, Canada.

Hutchinson, James P., M.D., Media, Pa.

Johnson, Robert W., A.B., M.D., 101 W. Franklin Street, Baltimore, Md.

Jonas, August F., M.D., 512 McCayne Building, Omaha, Neb.

Kammerer, Frederic, M.D., Lohn, Kehrsatz Ct., Berne, Switzerland.

Keen, W. W., A.M., M.D., LL.D., Hon. F.R.C.S. (Eng., Edin. and Ireland), Hon. Ph.D. (Upsala), Hon. ScD. (Jefferson and Harvard), Hon. F.A.C.S., The Touraine, 1520 Spruce Street, Philadelphia, Pa.

Lothrop, Howard Augustus, A.M., M.D., 101 Beacon Street, Boston, Mass.

McArthur, L. L., M.D., Ph.C.S., 122 S. Michigan Boulevard, Chicago, Ill.

Mac Laren, Murray, B.A., M.B., C.M., M.D., M.R.C.S. (Eng.) C.M.G., C.O.A., LL.D., M.P., 75 Coburg Street, St. John's, N. B., Canada.

Martin, Edward, A.M., M.D., LL.D., D.Sc., Media, Pa.

Mastin, Wm. M., M.D., LL.D., northeast corner Joachim and Conti Streets, Mobile, Ala.

Matas, Rudolph, M.D., LL.D., 2255 St. Charles Avenue, New Orleans, La.

Meyer, Willy, M.D., 700 Madison Avenue, New York City.

Monks, George H., A.B., M.D., M.R.C.S., 51 Commonwealth Avenue, Boston, Mass.

Mudd, Harvey G., M.D., Humboldt Building, St. Louis, Mo.
Murphy, Fred T., M.D., Penobscot Building, Detroit, Mich.
Murray, Francis W., M.D., 7 West Forty-third Street, New York, N. Y.

Neilson, Thomas R., M.D., 1937 Chestnut Street, Philadelphia, Pa.

Oliver, J. C., M.D., 628 Elm Street, Cincinnati, Ohio.

Parham, Frederick William, 3513 Prytania Street, New Orleans, La.
Parmenter, John, M.D., 519 Franklin Street, Buffalo, N. Y.
Pilcher, Lewis S., A.M., M.D., LL.D., 145 Gates Avenue, Brooklyn, New York.
Porter, Miles Fuller, M.A. M.D., 2326 Fairfield Avenue, Fort Wayne, Ind.

Rushmore, John D., M.D., 129 Montague Street, Brooklyn, N. Y.

Scudder, Charles L., A.B., Ph.B., M.D., 144 Commonwealth Avenue, Boston, Mass.
Shepherd, Francis J., M.D., LL.D., Hon. F.R.C.S. (Edin.), 152 Mansfield Street, Montreal, Canada.
Stillman, Stanley, M.D., Stanford University Hospital, San Francisco, Calif.
Summers, John Edward, M.D., Bankers' Reserve Life Building, Nineteenth and Douglas Streets, Omaha, Neb.

Taylor, William J., M.D., 1825 Pine Street, Philadelphia, Pa.

Vander Veer, A., A.M., M.D., Ph.D., LL.D., 28 Eagle Street, Albany, N. Y.
Vaughan, George Tully, M.D., LL.D., 1718 I Street, Washington, D.C.

Warren, J. Collins, M.D., LL.D., Hon. F.R.C.S. (Eng. and Edin.), 58 Beacon Street, Boston, Mass.
Watson, Francis S., A.B., M.D., South Dartmouth, Mass.
Weir, Robert F., A.M., M.D., Hon. F.R.C.S. (Eng.), 16 East Ninety-sixth Street, New York, N. Y.
Winslow, Randolph, A.B., M.D., A.M., LL.D., F.A.C.S., 1900 Mt. Royal Terrace, Baltimore, Md.
Woolsey, George, M.D., 117 East Thirty-sixth Street, New York City.

HONORARY FELLOWS OF THE ASSOCIATION

ELECTED

1907. Ballance, Sir Charles Alfred, K.C.M.G., C.B., M.V.O., M.B., M.S. (Lond.), F.R.C.S., 106 Harley Street, London, W. Consulting Surgeon to St. Thomas's Hospital and to the National Hospital for Paralysis and Epilepsy, Queen Square.

1919. Bastianelli, Raffaelle, Via dei Villini, 2, Rome, Italy; Consulting Surgeon, Hospitale Rome; Professor of Surgery, Royal University, Rome and Director of the Clinic.

1919. Bowlby, Sir Anthony A., K.C.B., K.C.M.G., K.C.V.O., F.R.C.S., D.S.M., (U. S. A.), Hon. F.A.C.S., 25 Manchester Square W., London, England. Consulting Surgeon to St. Bartholomew's Hospital and to Queen Alexandra's Military Hospital.

1922. Chutro, Dr. Pedro, University of Buenos Aires, Argentina. Professor of Surgery, (Retired), University of Buenos Aires; Honorary Professor, University of Madrid, Spain; Fellow, Royal Academy of Medicine of Spain, of the Académie de Médecine de Paris, of the Surgical Association of Buenos Aires, of the Société de chirurgie de Paris; Honorary Fellow, Association of Military Surgeons of the United States.

1919. Hartmann, Dr. Henri, Hon. F.R.C.S. (Eng.), 4 Place Malesherbes, xviie Paris, France. Professeur de Clinique Chirurgicale, Chirurgien de l'Hôtel Dieu. Membre de L'Académie de Médicine.

1920. Keen, W. W., A.M., M.D., LL.D., Hon. F.R.C.S. (Eng., Edin. and Ireland), Hon. Ph.D. (Upsala), Hon. Sc.D. (Jeff. and Harv.), Hon. F.A.C.S., 1520 Spruce Street, Philadelphia.

1918. Keogh, Sir Alfred, Hy., G.C.B., Hon. F.R.C.S., The Bungalow, East Grinstead, England.

1922. Leriche, Dr. René, 19 rue Jean Jacques Rousseau, Strasbourg, France. Professeur de Clinique Chirugicale à la Faculté de Médecine de Strasbourg.

ELECTED

1918. Makins, Sir George, G.C.M.G., C.B., LL.D. (Cambridge and Aberdeen), F.R.C.S., 33 Wilton Place, S. W. London, England.

1903. Mayo-Robson, Col. Sir Arthur, K.B.E., C.B., C.V.O., F.R.C.S., D.Sc., Broadoak, Seale, Surrey, Eng.; Consulting Surgeon, King Edward VII Memorial Hospital, Windsor, and Leeds Infirmary. Emeritus Professor of Surgery, University of Leeds; late Vice-President, Royal College of Surgeons and Hunterian Professor; Consulting Surgeon, M.E.F. (Dardanelles, Egypt, etc.), twice mentioned in dispatches; afterward Consulting Surgeon, Southern Council; member of Consulting Council, War Office; Consulting Surgeon, Croix Rouge Française; Chevalier Legion d'Honneur; Knight of the Order of St. John of Jerusalem.

1908. Moynihan, Sir, B. G. A., Bart., K.C.M.G., C.B., M.S. (Lond.), F.R.C.S., 33 Park Square, Leeds, England. Professor of Surgery, University of Leeds; Senior Surgeon, Leeds General Infirmary.

1907. Myles, Sir Thomas, M.D., F.R.C.S.I., 33 Merrion Square N., Dublin, Ireland.

1925. Power, Sir d'Arcy, K.B.E., M.A., M.B. (Oxon.), F.R.C.S. (Eng.), 10 A. Chandos Street, London, England. Consulting Surgeon, St. Bartholomew's Hospital; Vice-President, Bibliographical Society; formerly Vice-President, Royal College of Surgeons of England.

1912. Stiles, Sir Harold Jalland, K.B.E., LL.D., D.Sc., M.B., F.R.C.S. (Edin.), F.A.C.S., F.R.C.S.I., 9 Great Stuart Street, Edinburgh, Scotland. Regius Professor of Clinical Surgery, University of Edinburgh; President of the Royal College of Surgeons of Edinburgh; Surgeon to the Edinburgh Royal Infirmary and Consulting Surgeon to Chalmers Hospital and to the Royal Edinburgh Hospital for Sick Children, Edinburgh.

1924. Taylor, Sir William, K.B.E., C.B., M.D., LL.D., F.R.C.S.I., Hon. F.A.C.S., 47 Fitzwilliam Square, Dublin Ireland. Regius Professor of Surgery, Dublin University; Past President, Royal College of Surgeons in Ireland; Past President, Association of Surgeons of Great Britain and Ireland.

1918. Tuffier, Th., M.D., Hon. F.R.C.S. (Eng.), 42 Avenue Gabriel, Paris, France. Professeur agrégé de chirurgie; Membre de l'Académie de Médecine; Président de la section thérapeutique du Cancer, Hôpital cité du Midi, 48 boulevard de Clichy.

1920. Wallace, Sir Cuthbert, K.C.M.G., C.B., D.S.M. (U. S. A.), M.B., B.S. (London), F.R.C.S., (Eng.), 26 Upper Wimpole Street, W., London, Eng.; Surgeon to St. Thomas's Hospital, London; Dean of the Medical School.

1920. Walther, Charles, M.D., 49, rue de Courcelles, Paris, France. Professeur Agrégé à la Faculté de Médecine, Chirurgien Honoraire des Hôpitaux; Membre de l'Académie de Médecine.

DECEASED FELLOWS OF THE ASSOCIATION

Name	Date of death
Agnew, D. Hayes	March 22, 1892
Allen, Dudley Peter	January 6, 1915
Allis, Oscar Huntington	May 16, 1921
Annandale, Thomas	1907
Ashhurst, John, Jr.	July 7, 1900
*Atlee, John Light	October 1, 1885
Beckman, Emil Hessel	November 7, 1916
Benham, Silas Nelson	November 3, 1890
*von Bergmann, Ernst	Died since 1906 Meeting
*Bigelow, Henry Jacob	October 30, 1890
*Billings, John Shaw	March 11, 1913
*Billroth, Albert Christian Theodor	January 5, 1894
Bontecou, R. B.	1906–1907
Bosher, Lewis C.	September 12, 1920
Bottomley, John T.	December 17, 1925
Bradford, Edward Hickling	May 7, 1926
Briggs, William Thompson	June 13, 1894
Brinton, John Hill	March 18, 1908
Bristow, Algernon Thomas	March 26, 1913
Brock, Hugh W.	April 24, 1882
Bryant, Joseph Decatur	April 7, 1914
*Bryant, Thomas	1914–1915
Bull, William Tillinghast	February 22, 1909
Burrell, Herbert Leslie	1910
Byrd, William Andrew	August 14, 1887
Cabot, Arthur Tracy	November 4, 1912
Campbell, Henry Fraser	December 15, 1891
Cheever, David Williams	December 27, 1915
*Chiene, John	May 29, 1923
Cole, Charles Knox	February 27, 1920
Conner, Phineas Sanborn	March 26, 1909
Cunningham, Francis Deane	September 11, 1885
*Czerny, Prof. Dr. Vincenz	October 3, 1916

* Honorary.

Name	Date of death
Dandridge, Nathaniel P.	1911–1912
Davis, Gwilym G.	June 16, 1918
Davis, John Staige	July 18, 1885
Dawson, William Wirt	February 16, 1893
*Depage, Antoine	June 9, 1925
Dugas, Louis Alexander	October 19, 1884
Dunn, James Henry	June 16, 1904
Dunott, Thomas Justus	May 20, 1893
*Durham, Arthur Edward	May 7, 1895
Elliot, John Wheelock	September 17, 1925
*Erichsen, Sir John Eric	September 23, 1896
*von Esmarch, Friedrich	1907
Fenger, Christian	March 7, 1902
Ferguson, Alexander Hugh	October 20, 1911
Forbes, William Smith	December 17, 1905
Fowler, George Ryerson	February 6, 1906
*Friedrich, Paul Leopold	January 5, 1916
Gaston, James McFadden	November 17, 1903
Gerrish, Frederic Henry	September 8, 1920
Gerster, Arpad G.	March 11, 1923
*Gorgas, William C.	July 4, 1920
Greene, W. Warren	September 10, 1881
Gross, Samuel David	May 6, 1884
Gross, Samuel Weissel	April 16, 1889
Gunn, Moses	November 4, 1887
*Gussenbauer, Karl	June 19, 1903
Guthrie, George Washington	June 1, 1915
Halsted, William Stewart	September 7, 1922
Harrington, Francis B.	1914–1915
*Harrison, Reginald	1907
Harte, Richard H.	November 14, 1925
Hearn, W. Joseph	December 20, 1917
Hodge, H. Lenox	June 16, 1881
Hodgen, John T.	April 28, 1882
Homans, John	February 7, 1903
*Horsley, Sir Victor Alexander, C. B.	July 16, 1916
Horwitz, Orville	January 28, 1913
*Horwitz, Phineas J.	September 28, 1904

* Honorary.

Name	Date of death
Hotchkiss, Lucius Wales	April 11, 1926
Hughes, J. C.	August 10, 1881
*Humphrey, Sir George Murray	September 24, 1896
*Hunt, William	January 5, 1897
Jacobson, Nathan	September 16, 1913
Johnson, Alexander B.	September 4, 1917
Johnston, George Ben	December 20, 1916
Johnston, Christopher	October 11, 1891
King, Alfred	June 4, 1916
Kinloch, Robert Alexander	December 23, 1891
*Kocher, Theodor	July 27, 1917
Lane, Levi Cooper	February 18, 1902
*von Langenbeck, Bernhardt	September 30, 1887
Le Conte, Robert G.	1923
Levis, Richard J.	November 12, 1890
*Lister (Lord), Joseph	February 11, 1912
Little, James Lawrence	April 4, 1885
Lutz, Frank Joseph	March 24, 1916
McCann, James	1893
McCosh, Andrew J.	December 3, 1908
McGraw, Theodore A.	September 6, 1921
McGuire, Hunter	September 19, 1900
McMurtry, Lewis Samuel	February 1, 1924
McRea, Floyd W.	August 13, 1921
*MacCormac, Sir William	December 4, 1901
MacDonald, Willis Goss	December 30, 1910
*Macewen, Sir William	March 22, 1924
Mackenzie, Kenneth Alexander James	March 15, 1920
MacLaren, Archibald	October 12, 1924
MacMonagle, Beverly	May 22, 1912
*Maunoury, Gabriel	December, 1925
Markoe, Francis Hartman	1907
Marks, Solon	1916
Mastin, Claudius Henry	October 3, 1898
Mears, J. Ewing	May 28, 1919
Michael, J. Edwin	December 7, 1895
*Mikulicz-Radecki, John V.	June 17, 1905
Miles, Albert B.	August 5, 1894
Millard, Perry H.	February 1, 1897

* Honorary.

Name	Date of death
Miller, Truman W.	May 31, 1900
Mixter, Samuel Jason	January 19, 1926
*Moore, Edward Mott	March 3, 1902
Moore, James E.	November 2, 1918
Mudd, Henry Hodgen	November 20, 1899
Mumford, James Gregory	October 18, 1914
Munro, John C.	December 6, 1910
Murphy, John B.	August 11, 1916
de Nancrède, C. B. G.	April 12, 1921
Nichols, Edward Hall	July 6, 1922
Norris, Basil E.	October, 1895
*von Nussbaum, J. N.	October 31, 1890
Ochsner, A.J.	July 25, 1925
*Ollier, Leopold	1900–1901
Oviatt, Charles W.	October 31, 1912
Owens, John E.	December 21, 1922
*Paget, Sir James	December 30, 1899
Pancoast, William H.	January 5, 1897
Park, Roswell	February 15, 1914
*Parker, Willard	April 25, 1884
Parkes, Charles T.	March 28, 1891
Parkhill, Clayton	January 16, 1902
*Péan, Jules E.	January 30, 1898
Peck, Washington F.	December 30, 1891
Pilcher, Paul Monroe	January 4, 1917
Porter, Charles Burnham	May 21, 1909
Porter, William Gibbs	January 30, 1906
Powers, Charles A.	December 23, 1922
*Pozzi, Samuel	1918
Prewitt, Theodore F.	October 17, 1904
Prince, David	December 19, 1889
Ransohoff, Joseph	March 10, 1921
Reed, Thomas Baird	April 1, 1891
Richardson, Maurice Howe	July 31, 1912
Richardson, Tobias Gibson	May 26, 1892
Roberts, John B.	November 28, 1924
Rodman, William Louis	March 8, 1915
Ross, George Gorgas	December 27, 1922

* Honorary.

Name	Date of death
Sabine, Thomas Taunton	August 23, 1888
Sayre, Lewis A.	1900–1901
*Schede, Max	1902
Senn, Nicholas	January 2, 1908
Sherman, Harry M.	1921.
*Sims, J. Marion	November 13, 1883
*Smith, Stephen	August 26, 1922
Souchon, Edmond	August 5, 1924
Squire, Truman Hoffman	November 27, 1889
Stewart, Francis T.	February 4, 1920
*Terrier, Louis Felix	1908
*Thiersch, Karl	April 28, 1895
Thompson, Joseph Ford	February 1, 1917
Thompson, Joseph W.	March 30, 1886
*Thomson, Alexis	March 4, 1924
Thomson, William	August 3, 1907
Tiffany, Louis McLane	October 23, 1916
Varick, Theodore Romeyn	November 23, 1887
*Verneuil, Aristide A. S.	June 11, 1895
*von Volkmann, Richard	November 28, 1889
Walker, Edward W.	October 7, 1925
Watson, Beriah Andrew	December 22, 1892
Weeks, Stephen Holmes	September 1, 1909
Weist, Jacob Rowland	May 14, 1900
*Wells, Sir Thomas Spencer	January 31, 1897
Wharton, Henry R.	December 3, 1925
White, James William	April 24, 1916
Wight, Jarvis S.	November 16, 1901
Willard, DeForest	October 14, 1910
Wood, James R.	May 4, 1882
*Yandell, David Wendell	May 2, 1898

* Honorary

ADDRESS OF THE PRESIDENT

THE PSYCHOLOGY OF THE SICK MAN

By JOHN H. GIBBON, M.D.
PHILADELPHIA

A YOUTH in love feels that he is experiencing sensations which no other member of his sex ever felt, and finds the language altogether inadequate to express his thoughts. A contemplation of my present situation, to which you have elected me with a friendly disregard of my qualifications, produces sensations quite similar to the adolescent suffering his first reaction to the inoculation of Cupid's dart. My heart is so full of appreciation and gratitude that I am rendered absolutely aphasic and, like the lovesick swain, I fall back on time-worn phrases and simply say, I thank you. I know that my predecessors in this chair have felt as I do at this moment, and, in order that the future occupants may suffer and enjoy the same sensations, let us hold to the traditions and standards which have made the American Surgical Association the best surgical society in the world.

In choosing for my subject the "Psychology of the Sick Man," I purpose to avail myself of one of the privileges of the office and indulge in broad philosophic wanderings over the realm of medicine as some of my predecessors have done, and I think to advantage.

I should first like to offer as a proposition that the advancement of the art of surgery will not come with the invasion of new anatomic fields nor with the further perfection of technic, except in the field of anesthesia; but will come with increased knowledge of the cause and prevention of disease, with the advancement of diagnostic methods, with the exercise of better surgical judgment and with a broader knowledge of general medicine. This may

prove a poor prophecy, but you will agree that the sources from which I have indicated advancement might come, certainly represent fields in which we particularly need to work.

It is a trite statement that the more a surgeon knows of general medicine the wiser surgeon he is; but with our present-day methods of education and the arrangement of our intern services and apprenticeships, the foundation on which the young surgeon has to build is too narrow. It would be far better for the young man if he could forget during this preparatory period that he is to become a surgeon and devote himself to the acquirement of as broad a knowledge of medicine as possible. It must be understood at the outset that I am looking on surgery as an art, and the man who practises it as one upon whom his fellow-man can call in time of need and expect to find a practical man, possessing not only knowledge but wisdom. Who of us, if sick, would choose as his physician a scientist? Who would not choose the man best versed in the art of medicine, one who utilizes all that science can give him and who is capable of applying his knowledge in a practical way and who has had a broad experience in the practice of his art? I would not be understood to decry scientific research, since modern surgery owes to it its being; but the scientist is a poor physician largely because of his limited field of wisdom and experience.

It is of the psychology of the patient, however, that I want to speak at some length, as I believe it is a matter to which the average surgeon pays little attention and this little subconsciously. It is not exactly a neglected subject; for instance, much of the good to be derived from Crile's "anoci-association" is due to the consideration given to the mental state of the patient. Every surgeon needs to consider the patient's attitude toward his ailment, and should be able to distinguish to a certain extent the imaginary from the real symptoms. He should know how to help the patient rid himself of those which are not real and also how to avoid inspiring or augmenting them.

The layman today likes to think he knows something about disease and its treatment, and is very apt to think he is in a position to decide the type of treatment which his peculiar kind of malady requires. The man who is sick, or who thinks he is sick, is most susceptible to suggestion, is keen to put his own interpretation on a chance word, an expression of the face or a

single laboratory finding; with the result that he is, at least mentally, either better or worse. Nothing is more impressionable than the mind of a sick man, and it is the realization of this fact and the use of it to their own ends that has caused the quacks of all ages to prosper. It is to the exploitation of this human weakness that Christian Science, osteopathy and chiropracty owe their success.

Every practitioner of medicine should understand something about psychology, about hysteria and about psychotherapy. He should know what havoc imagination can work, what feeds it and how it can be suppressed. Many a physician or surgeon, in taking a history by suggestive questioning, by explaining to the patient under the idea that he is being perfectly frank and honest the result of certain laboratory studies, and by giving in detail the necessary treatment, may very easily be adding to his patient's suffering. Many of our modern methods of study have this effect. The various commercial societies and companies which have sprung up all over the country, urging the healthy as well as the sick to subject themselves, often through the medium of the mail, to a thorough examination of all their functions at stated intervals and then putting the results into the patient's own hands, are doing much more harm than good. Such examinations by a conscientious and capable physician, who can also study the mental attitude of the patient, would be an entirely different thing and produce only good results. I am always sorry for the poor patient who turns up with his *x*-ray plates, his history and the reports from the various laboratories in his hands, and then tries to make his symptoms correspond to them. When one tries to reassure him he comes back with, "Well, how do you explain the shape and position of my colon in these plates?" or, "But the roentgenologist says that I have chronic appendicitis," or, "How can I get rid of those streptococci in my bile? You ought to see my bile." How much better off and how much easier to cure is the poor human derelict who comes into the ward not knowing a thing about himself, or the intelligent man who puts himself confidently in the hands of his physician and does not want to know all about the "findings." Under the mistaken idea of honesty many of our specialists are making neurasthenics of their patients, and I sometimes think that behind a good deal of their frankness is the desire to impress the patient with their

profound knowledge and thoroughness. Pope must have had in mind the "Malade Imaginaire" when he wrote:

> "A little learning is a dangerous thing;
> Drink deep, or taste not the Pierian spring,
> There shallow draughts intoxicate the brain,
> And drinking largely sobers us again."

and Donne, I am sure, was thinking of the neurasthenic when he said, "Who are a little wise, the best fools be."

Much of the sex hygiene taught in schools is presented in such a way as to do enough harm in certain cases to offset all the good it does in others. I can sympathize with the poor mother who wrote as follows: "Dear Teacher: Don't learn Mary no more about her insides. Its rude and it don't do no good." It is just as important for a surgeon to realize the possibilities of suggestion as it is for any other practitioner of medicine.

That well-trained physician and neurologist of Guy's Hospital, Arthur F. Hurst says: "I believe that the most common source of suggestion of hysterical symptoms is some organic disease or injury, the symptoms resulting from which are perpetuated or aggravated by autosuggestion, sometimes with the help of the unconscious heterosuggestion produced by the questions and the treatment recommended by the physician, if he does not recognize the true nature of the condition. When thus produced, hysterical symptoms always simulate the organic symptoms which preceded them more or less closely. In many cases, when a certain degree of improvement has occurred in the original organic condition, a mixture of an organic basis with a superimposed hysterical element is present. I believe that this is a much more common event than is generally supposed, and that every organic incapacity tends to suggest a greater incapacity."

A good surgeon must be a good diagnostician and should not operate on someone else's diagnosis. If he is not capable of diagnosing the diseases he treats, he should not treat them.

In the training of the student and young surgeon too much stress is laid on surgical technic and too little on the pathology and natural history of disease and on diagnosis. Diagnosis by exclusion is an excellent plan, but we surgeons too often make this a physical exclusion. In other words, in order to make a diagnosis of a neurosis it should not be necessary to remove first

the appendix, then the gall-bladder and then the colon. This method of reaching a diagnosis, which neurologists like to think of as being the surgical method, not only is of no value, but by the time the correct diagnosis is made the patient is often beyond hope or he becomes one of the much-lauded cures of Christian Science or osteopathy.

Just to illustrate that I am not indulging in exaggeration, I should like to refer to the case of a nervous but fat and healthy-looking young man who for a number of years had been in the hands of different internists, who treated him largely for mucous colitis. He was sent by his physician, who was at the end of his string, to a surgeon with a diagnosis of chronic appendicitis. This was in 1919. There was little evidence of appendicitis, but there was present an incomplete hernia. This was operated upon and the appendix removed through the sac. During the next two and a half years this patient continued to complain of vague and indefinite upper abdominal symptoms and was examined and treated by a great many physicians. The surgeon who operated upon him in the first instance doubted the existence of a lesion; but the patient had been to a large sanatorium, where his *x*-ray plates were explained to him as showing undoubted disease of the gall-bladder, and he was told that operation was indicated. He was then carefully studied by a very capable gastroenterologist, who also advised operation in spite of his undoubted neurotic symptoms. His upper abdomen was opened and the gall-bladder and ducts, as well as the stomach and duodenum, found to be absolutely normal. These negative findings instead of being a comfort to the patient only disturbed him the more, and he sought advice of another surgeon eighteen months later. This surgeon operated and found his gall-bladder and duodenum adherent, but no other lesion, and removed his gall-bladder. This was in May, 1923. He was "cured" for a brief season, but his symptoms returned, and later another surgeon operated upon him for "adhesions," which were separated and the colon fixed between the duodenum and the liver. This did not relieve the situation, however, and within a year, on the advice of a distinguished internist who thought that he might have a duodenal ulcer, in spite of the fact that he had had three previous operations by experienced surgeons, he was again operated upon. No ulcer was found, but the distal portion of the stomach was removed.

At the present time he is being treated by a specialist for infection of the gums. Every one of the four surgeons who operated upon this man was a Fellow of this Association, myself, I am ashamed to say, one of them.

This is not an unusual story, but I do not think we learn the lesson which these cases teach. We study our physical results, we take pains to eliminate or lessen the operative risks, but would it not be well to go into the psychologic and gastroenterologic clinics occasionally and see some of the results of our mistakes in diagnosis and treatment? Would it not be well to have the neurologist see some of these cases before rather than after operation? Oh, I am sure that I am not wide of the mark in insisting that surgical results can be improved by a familiarity on the part of the surgeon with the various neuroses, psychoses and hysterias. I realize that many useless and harmless operations, such as the removal of the colon, are being done on the insane at the behest of a few unbalanced psychiatrists with the idea of actually curing the mental disease. No insane patient is ever cured of insanity by a surgical operation done on his abdominal or on her pelvic viscera, and the neurotic and hysterical patient is invariably made worse ultimately by such operations, which very rightly bring discredit on surgery. It, of course, goes without saying, that the insane patient and the neurasthenic who has a real surgical lesion should have exactly the same treatment given a mentally normal person.

In every contact with the sick the surgeon must constantly bear in mind the effect his words and actions may have on the patient. Interns and nurses need to have this strongly impressed on them, and here example is better than precept: A perfectly normal woman was recently troubled, upset and disturbed, after a colon resection for cancer, by her physician who said, "Now it is all out, and if you don't get an obstruction you will be all right." Of course, with every subsequent "gas pain" she thought that the obstruction had arrived. A surgeon should inspire confidence, assurance and faith, and must be prepared to justify them with a sympathetic and conscientious exhibition of ability. A visit made to a patient after an operation which does not leave him cheered, comforted and more hopeful, had better never been made. It should always be realized that an indiscreet word, an

anxious look or a lugubrious manner will leave the patient depressed, worried and full of fear. In the practice of surgery wisdom is as necessary as knowledge and not so easily acquired. "Knowledge comes, but wisdom lingers."

> "Knowledge is proud that he has learned so much;
> Wisdom is humble that he knows no more."

Ian Hay, in a recent address to the students of Guy's Hospital, talked on "The Human Touch," and said among other good things, "Tell the patient something that will keep his imagination from soaring into the regions of unhealthy speculation." I would only add that we should avoid saying or doing anything that would turn the mind into these unpleasant channels. We surgeons every day have to tell poor, suffering, nervous humans unpleasant and disturbing facts; but let us tell them as we would have them told to us. Lying is not necessary and is a poor policy, if for no other reason than that it sooner or later is discovered and destroys confidence. No rule can be laid down, but the patient's mental attitude and the effect upon it by what is said, must be considered.

During convalescence coöperation on the part of the patient is most helpful and sometimes an absolutely essential element in restoring health and function. Cheering friends tell our abdominal cases that they will not "get over the effects of the operation for a year," and some of them will try their best to carry out the program. Tell a patient after a fracture of the leg that he will be lame for six months, and whether he needs to or not he will limp for the allotted time. Limps in the absence of shortening or fixation are nearly all hysterical and can be readily overcome.

Not only should the surgeon know something of the neuroses, but he should be able to recognize the various manifestations of hysteria and realize their close resemblance to the symptoms of real surgical lesions. We have all known patients to undergo repeated operations for hysterical vomiting and for hysterical intestinal obstruction and then to be disappointed because further operations were refused.

In the field of traumatic and industrial surgery something more is required than a knowledge of surgery. The surgeon in this field must be able to distinguish the real sufferer, the hysterical

sufferer and the malingerer, and the last is the most infrequent and the second much more common than is generally believed. Even in many cases involving compensation or litigation the apparent malingerer is not a malingerer at all, but suffering from hysteria, the result of suggestion at the hands of friends, of fellow-workers, of his legal adviser and of partisan medical experts. This fact is pretty generally known, but do we realize how often it applies to cases in which there is no question of litigation? We must get over the idea that hysteria will always produce the physical stigmata of Charcot. Babinski and others have shown the fallacy of such an idea and that a perfectly normal person can suffer from hysteria. We surgeons can, in our own experience, amply illustrate this fact. The hysterical incapacities after operation and injury are everyday occurrences, and, although we may not designate them as hysterical, we prevent and cure them by suggestion and persuasion, and in doing so we are practising psychotherapy, although we may not realize it.

I shall always feel indebted to Sir William Osler for suggesting a visit during the World War to a neuropathic hospital in charge of Colonel Hurst, for here I learned in one morning a great deal about hysterical spastic palsy which has proved of great value since. There are hundreds of men, women and children wearing apparatus or submitting themselves to repeated operations for this condition, who could be easily cured by suggestion. These are the patients who largely represent the cures accomplished at Lourdes, at Ste. Anne de Beaupré and at other shrines and by the bone-setters and the Christian Scientists. That these poor people get into this apparently hopeless condition is due largely to the fact that the nature of their affliction is never properly diagnosed or because we do not know how to prevent or cure it. I saw many cases of perfectly honest British "Tommies" who had suffered for months, and some for years, from these palsies for which some of them had been discharged from the army as incurable, cured in ten minutes by psychotherapy. A good example is that of a sergeant who had had a "through-and-through" wound of the forearm a number of months previously, and who, since his arm was taken off the splint, had held his fingers tightly flexed on the palm until the growing nails had made ulcers. This man in five minutes was completely extending his fingers, together and

individually, much to his own astonishment and joy. Another case in civil life, which illustrates very well what I want to say, was that of a young man who was sent to the Jefferson Hospital, Philadelphia, from one of the towns in northern Pennsylvania. He had a fracture of the clavicle which a surgeon had wired, and following the operation the patient had never been able to abduct the arm more than a few inches from the chest wall. He was supposed to have an ankylosis of the shoulder and the *x*-ray plates were thought to show certain changes in the bones and joint which would account for the disability. As massage, electricity and exercise had accomplished nothing after months of use, operation was advised. From the general muscular rigidity whenever the patient attempted abduction, and from the fact that when this was overcome by persuasion certain movements could be easily carried out, a diagnosis of hysterical spastic palsy was made, and in ten minutes this boy was carrying the arm up in full extension over his head. Massage, electricity, apparatus, operation—all have their place, but are harmful in this condition since they only prolong it, and because it can be quickly cured by psychotherapy.

An important point is that patients should not be allowed to get into this condition, and it is easily prevented. In this connection I would advise all young surgeons to read Col. Hurst's article in the Osler Memorial volume on "What the War Has Taught Us About Hysteria."

One who doubts the effect of mind over matter should read Klauder's paper (*Jour. Am. Med. Assn.*, November 28, 1925) on the "Cutaneous Neuroses," in which he shows among other interesting tests that, by suggestion, blisters can be made with postage stamps.

The diagnostic habit needs to be cultivated by the surgeon, and the young man needs to be taught that there is something more in the art of surgery than operative skill and technic. The link between surgery and psychology is too important to be neglected. Lawrence Sterne said of his teachers at Cambridge that they "were men of reading who thought that 'wisdom can speak in no other language than Latin and Greek,'" and I sometimes think there are too many practitioners of medicine who, in making a diagnosis, depend too much on the laboratory findings

and fail to recognize many perfectly patent signs and symptoms which one experienced in the art sees at a glance. The wise practitioner knows his Latin and Greek of the laboratories and uses them, but he does not start or stop with them.

> "Canst thou not minister to a mind diseas'd,
> Pluck from the memory a rooted sorrow,
> Raze out the written troubles of the brain,
> And with some sweet oblivious antidote
> Cleanse the stuff'd bosom of that perilous stuff
> Which weighs upon the heart?"

THE OCCURRENCE AND MANAGEMENT OF GASTRO-JEJUNAL ULCER

By DONALD C. BALFOUR, M.D.
ROCHESTER, MINN.

Recurrence of ulceration may follow any operation for peptic ulcer, including partial gastrectomy. Many factors are worthy of consideration as the cause of these recurrences, but in the individual case it is seldom that such possible causes can be established. It is, however, becoming increasingly apparent that a certain type of individual, the hypertonic, is particularly prone to recurrence of ulceration. The tendency of such recurrent lesions to localize at, or near, the site of a gastro-enterostomy is well known, and their clinical recognition is one of the outstanding advances in diagnosis in recent years. The diagnosis of these gastro-jejunal ulcers by roentgen-ray is not as positive as it is of other lesions of the stomach or duodenum, and a negative report is of relatively minor value in a case with a clear-cut clinical history. The course of gastro-jejunal ulcer is more definitely progressive (particularly in the disability of the patient) than that of a primary ulcer, and it is subject to the same complications, except that malignant degeneration has not been reported. The treatment of gastro-jejunal ulcer is surgical, and it is this phase of the subject I wish to discuss.

This presentation is based on a series of 270 cases of gastro-jejunal ulcer operated on in the Mayo Clinic. In 139 of these the original operation for ulcer had been performed in the Clinic, and in 131 it had been performed elsewhere. Gastro-enterostomy has been performed for peptic ulcer more than 8600 times in the Clinic, so that the total percentage of gastro-jejunal ulcers is 1.6. This percentage is approximately the same as that reported from many of the larger clinics, particularly those of Moynihan, Sherren, and Walton. The average age of the patients with gastro-jejunal

ulcer was forty-two, the oldest being seventy-four, and the youngest fifteen.

The incidence of males and females is interesting, since it is a fact that gastro-jejunal ulcer occurs rarely in women. In this series there were 248 males and 22 females, a ratio of 10 to 1, while the ratio of primary peptic ulcer encountered in men and women is 3.5 to 1.

Again it is shown that the likelihood of gastro-jejunal ulcer following gastro-enterostomy for duodenal ulcer is considerably greater than following gastro-enterostomy for gastric ulcer. Because of incomplete information as to those patients operated on elsewhere, we can only judge the relative liability of our own cases. Of those cases in which gastro-enterostomy was performed at the Clinic gastro-jejunal ulcer followed an operation for duodenal ulcer in 130 and followed operation for gastric ulcer in only 9. While this ratio is 15 to 1, the ratio between the number of cases in which gastro-enterostomy was performed for duodenal ulcer and those in which it was performed for gastric ulcer is 7 to 1.

The acid values of the gastric contents in cases of gastro-jejunal ulcer as compared to the acid values before gastro-enterostomy are usually, but not always, of significance. In 40 per cent of this series of cases of gastro-jejunal ulcer the free hydrochloric acid was either increased or very slightly reduced by the gastro-enterostomy; but in 40 per cent there was a marked reduction, and in 20 per cent the free hydrochloric acid was reduced to zero. The fact that there was no free hydrochloric acid in one-fifth of the cases of gastro-jejunal ulcer in which repeated and fractional examinations of the gastric contents were made disproves the assumption that achlorhydria following the primary operation affords protection against later ulceration.

Perforation, either chronic, subacute or acute, characterizes all gastro-jejunal ulcers and involves first in frequency the mesocolon and adjacent peritoneal folds, next the colon, and third the abdominal wall.

The average length of time between the operation for the primary ulcer and that for the gastro-jejunal ulcer was four and a half years in this series. In 56.7 per cent of the cases symptoms suggestive of recurrence appeared within the first year. There were, however, a number of cases in which symptoms did not recur for a considerably longer period, in one instance about twelve years. Such long periods before the recurrence are exceptional.

The indications for management are clear. Experience emphatically teaches that when the symptoms are those of recurring ulceration, when the roentgenogram is positive, and when relief from symptoms cannot be promptly attained and maintained by medical treatment, early operation is the safest and most satisfactory method of management.

The conditions found at exploration will determine the best procedure to be followed. It may always be assumed that a well-placed anastomosis has brought about the healing of a duodenal ulcer. If such healing has taken place without producing any obstruction at the pylorus, and if examination of the duodenum and pyloric end of the stomach seems to show that it will maintain adequate drainage of the stomach, the simplest and most rational procedure to employ is the disconnection of the anastomosis and the excision of the gastro-jejunal ulcer. When such an operation can be performed, it must be looked on as the operation of choice, and it has the very great merit of its conservatism. The patient should, in his habits of living following operation, endeavor to prevent possible reactivation of the ulcer, and if such reactivation should occur, he should be informed of the advisability of radical operation.

It is more often true, however, that although the original ulcer has healed, it has healed with such extensive scarring, or so much deformity has resulted from previous operations on the pylorus, that the latter is incapable of carrying on its normal function, and an operation as simple as the disconnection of the anastomosis is, therefore, unwise. When this simple operation cannot be performed the operation of election, from the standpoint of immediate and late results, is the disconnection of the anastomosis with excision of the ulcer and partial gastrectomy.

When either the simple or more radical operation is contraindicated, one of the many other procedures may be substituted. Of such procedures the first in merit is the disconnection of the anastomosis, excision of the ulcer, and an operation at the pylorus to provide for adequate drainage. When satisfactory drainage has been secured the results often have been completely satisfactory, but they are also uncertain; and in my opinion the indications for this procedure are becoming less and less frequent. An important point in the technic of such operations, whether they be of the pyloroplasty type or whether a Billroth I (removing

only a small part of the pyloric end of the stomach), is to fix the reconstructed pyloric outlet to the right of the median line by a stay suture between the anterior wall of the stomach and the round ligament of the liver, as suggested by W. J. Mayo.

Other procedures which are more rarely performed because of their uncertain results, and only under most unusual circumstances, are: (1) Excision of the ulcer and a plastic operation on the gastro-enteric stoma when the ulcer is small and the stoma large, well situated, and free from mesocolic attachments; (2) a second gastro-enterostomy when technical difficulties apparently prohibit the mobilization of the anastomosis; (3) the disconnection of the anastomosis with excision of the ulcer and a second gastro-enterostomy in cases in which the original gastro-enteric stoma is so small or so placed that it is quite incapable of functioning; (4) partial gastric exclusion after the method of Devine, when the gastro-enteric anastomosis seems capable of carrying on function, and (5) jejunostomy as recommended by Moynihan. All these procedures are uncertain in their results and are only to be considered when other methods are contraindicated.

It can safely be said, therefore, that with the exception of the simple disconnection of the anastomosis the best surgical treatment for gastro-jejunal ulcer is partial gastrectomy and that the various operations which have been described are to be employed only when there are definite contraindications to resection of the stomach.

Up to April, 1926, at the Mayo Clinic we have performed partial gastrectomy for uncomplicated gastro-jejunal ulcer in 89 cases with death in 3, a mortality rate of 3.37 per cent. This mortality rate is not excessive in view of the frequently formidable nature of the operation, the condition of the patient, and the excellent results which follow the operation. The most common and unfortunately the most serious complication of gastro-jejunal ulcer is the formation of a gastro-jejuno-colic fistula. Such a complication adds to the difficulty and risk of the operation. It is actually an unnecessary complication since there is always evidence of the ulcer long enough before the development of the fistula for adequate surgical treatment to be instituted.

The success of partial gastrectomy for gastro-jejunal ulcer does not depend alone on its technical perfection. In the first place the value of observation in the hospital before operation cannot

be overstressed. In a previous article I have pointed out the advantages of preoperative treatment in this type of case.[1] Because of long-continued dietary restrictions, pain, loss of sleep, and worry, many of the patients exhibit serious malnutrition. The disability in some cases is complete, and these patients are greatly benefited by a few days in the hospital, during which time they rest in bed and receive a bland diet and fluids in sufficient quantity to overcome the dehydration. Such measures are particularly beneficial when recent hemorrhage, exacerbation of inflammatory process, or retention have occurred. Not only is the general condition of the patient improved, but the activity of the lesion itself and the inflammatory reaction associated with it are reduced.

In the case of an uncomplicated gastro-jejunal ulcer partial gastrectomy can usually be conducted in a very precise manner: each step in the operation should be completed before subsequent steps are undertaken. It is hardly necessary to say that one should make as complete an exploration of the abdomen as is possible; although, when several operations have already been performed, detailed exploration involving extensive dissection is unwise because of the time consumed and the trauma inflicted.

The first step in the operation is the mobilization of the stomach. It is usually advisable to approach the anterior wall of the stomach and free it from its usual attachments to the abdominal wall in the region of former incisions and to the liver. The pylorus is then exposed and also the duodenum, whenever advisable, to determine the extent of pathologic changes there. The examination of the stomach should be most thorough in order to exclude the presence of a gastric ulcer at a higher point than the line on which the stomach will be resected. The colon and the great omentum are then mobilized and the gastro-enteric anastomosis brought into view. The anastomosis is inspected for ulceration or for signs of irritation. These can usually be immediately detected and are most commonly seen in the anterior aspect of the anastomosis. In the majority of cases the site of the inflammatory process is at either the proximal or the distal extremity of the anastomosis. The mesocolon is invariably involved in this inflam-

[1] Balfour, D. C.: The Value of Coöperation between Internist and Surgeon in the Management of Complicated Gastric Conditions, with Some Remarks on Partial Gastrectomy, Jour. Am. Med. Assn., 1925, lxxxiv, 876–879.

matory process, and in many cases the inflammatory thickening in the mesocolon and gastro-colic omentum completely obscures the lesion (Fig. 1). The separation of the anastomosis and the excision of the ulcer are very much facilitated if the entire anastomosis can be satisfactorily mobilized before one attempts to separate the jejunum from the stomach. An ulcer in the line of anastomosis is characteristically of the perforating type, the base

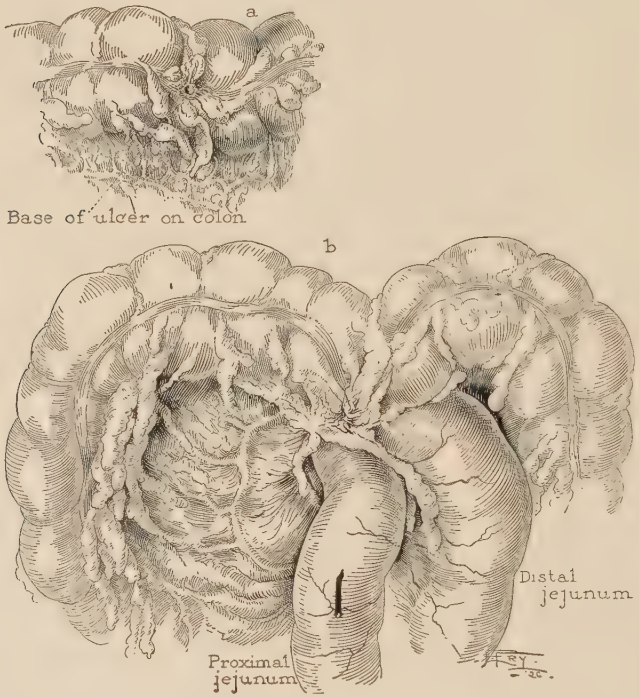


Fig. 1.—Illustrating how inflammation of the surrounding structures may obscure a gastro-jejunal ulcer.

of the lesion often being found in the surrounding fat of the mesocolon or gastro-colic omentum, the mesentery of the jejunum, or the wall of the colon. A very useful method which applies, apparently, to all cases, particularly to the more difficult ones, is to expose the posterior wall of the stomach at some point near the anastomosis and by finger dissection to free the posterior wall of the stomach from its mesocolic attachments. This makes it more

feasible to lift the mesocolon from the stomach anteriorly by blunt dissection until the edge of the mesocolon can be dissected by knife from its attachment (usually the anastomosis) (Fig. 2). After the mesocolon has been separated from the stomach, the segment of stomach is drawn down through the opening in the mesocolon and a rubber covered clamp placed across this segment of stomach 4 or 5 cm. from the site of the anastomosis. Likewise, the jejunum having been mobilized by the dissection, a rubber-covered clamp is placed across its two limbs and a gauze pack placed behind the anastomosis (Fig. 3).

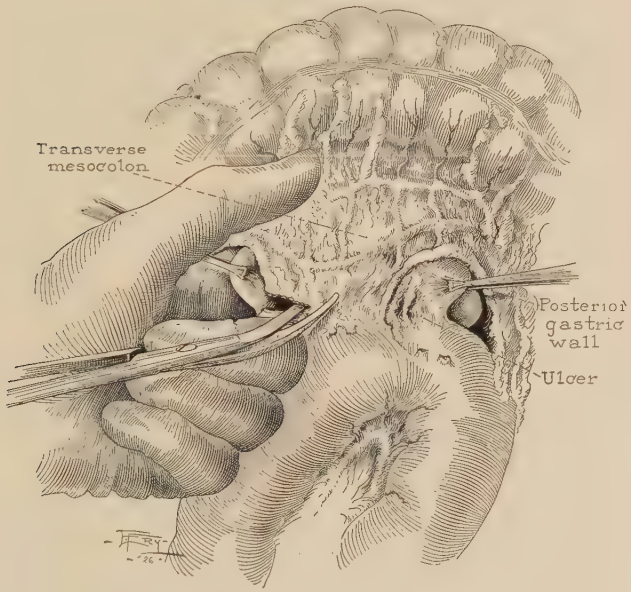


FIG. 2.—Method of freeing the mesocolon from its attachments.

A very useful way of demonstrating the exact site and size of the ulceration is to divide the stomach 2.5 cm. from the anastomosis. The resulting cuff of stomach can then be turned back on itself and the whole line of the anastomosis is seen before it is damaged. If the ulcer is in the suture line or close to it, either in the stomach or the jejunum, the anastomosis with the cuff of the stomach attached to it can be excised, the ulcer being excised with this cuff (Fig. 4). If the ulcer is in the jejunum at some dis-

tance from the anastomosis it is not necessary to excise it, since healing apparently takes place promptly when the anastomosis is

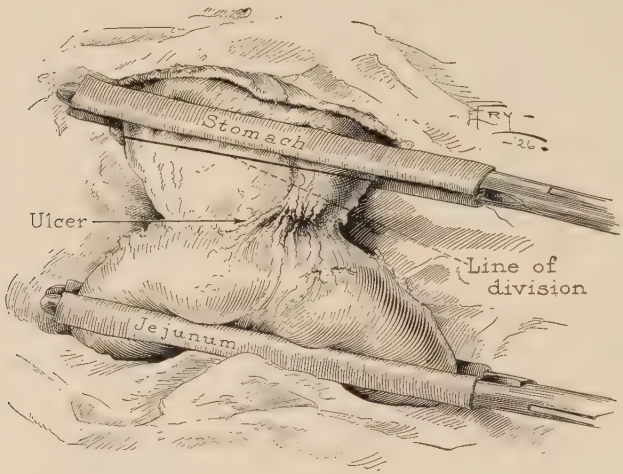


FIG. 3.—Application of clamps to stomach and jejunum on each side of the anastomotic ulcer.

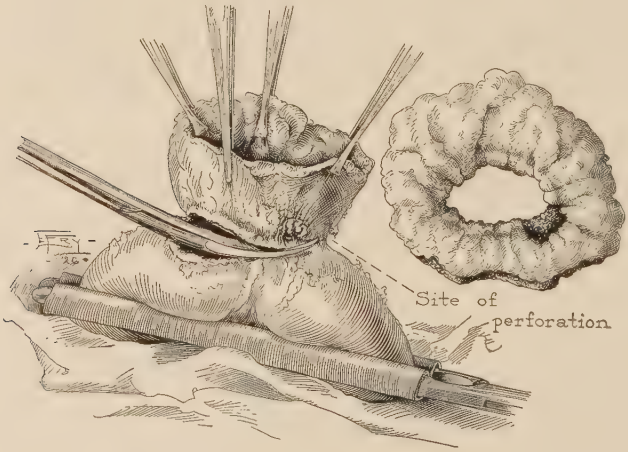


FIG. 4.—Excision of gastro-jejunal cuff containing the ulcer.

disconnected, and if the new union is made distal to the lesion. The defect in the jejunum is then closed in a transverse direction with chromic catgut. As a matter of convenience the opening in

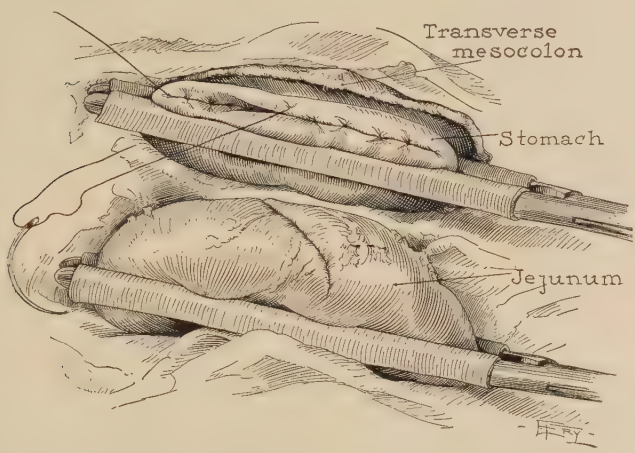


FIG. 5.—Closure of the stomach preparatory to resection.

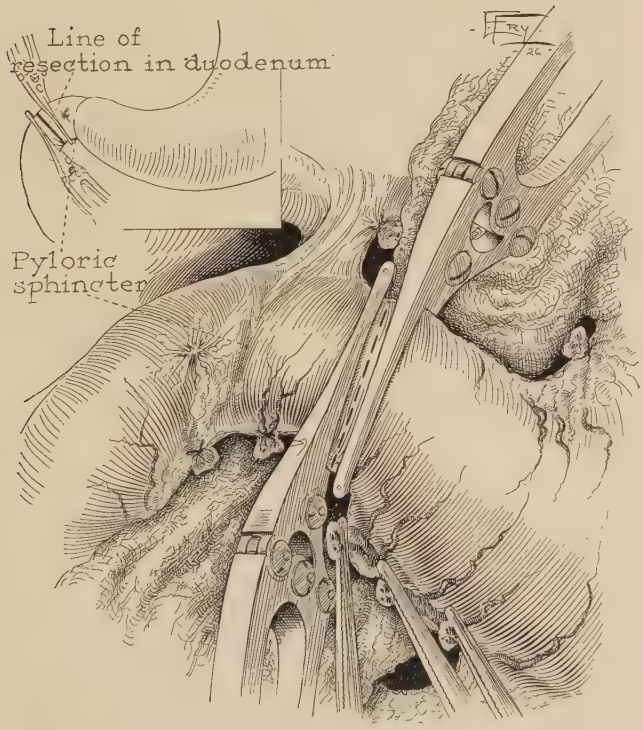


FIG. 6.—Application of clamps for resection of stomach.

the stomach is closed (Fig. 5) with a running suture of chromic catgut and the stomach is resected in the usual way. It is important in the resection to avoid extensively scarred areas of the duodenum by beginning the resection sufficiently far above the pylorus for the pyloric stump to be easily closed (Fig. 6). The stomach is then mobilized by a division of the gastro-colic and the gastro-

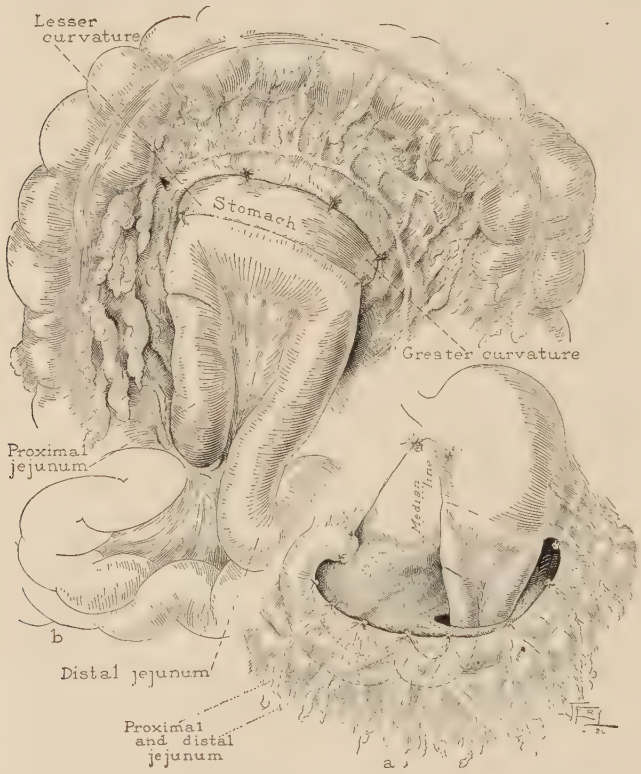


FIG. 7.—Completion of posterior end-to-side anastomosis.

hepatic omentum to a point high enough to permit of the removal of a sufficiently large portion of the stomach. A rubber-covered clamp is then placed across the stomach, parallel to the longitudinal axis of the body, 2.5 cm. above the site of the division. It is an important feature at this stage to place the clamp lightly enough not to injure the mucosa. I have had 1 case in which I am

convinced that the clamp caused enough damage to the mucosa to prevent healing, and a large ulcer formed which subsequently perforated against the diaphragm and necessitated an operation for the removal of this recurring ulcer.

If the resection is not too extensive and the remaining segment of the stomach is not fixed by adhesions, a posterior end-to-side

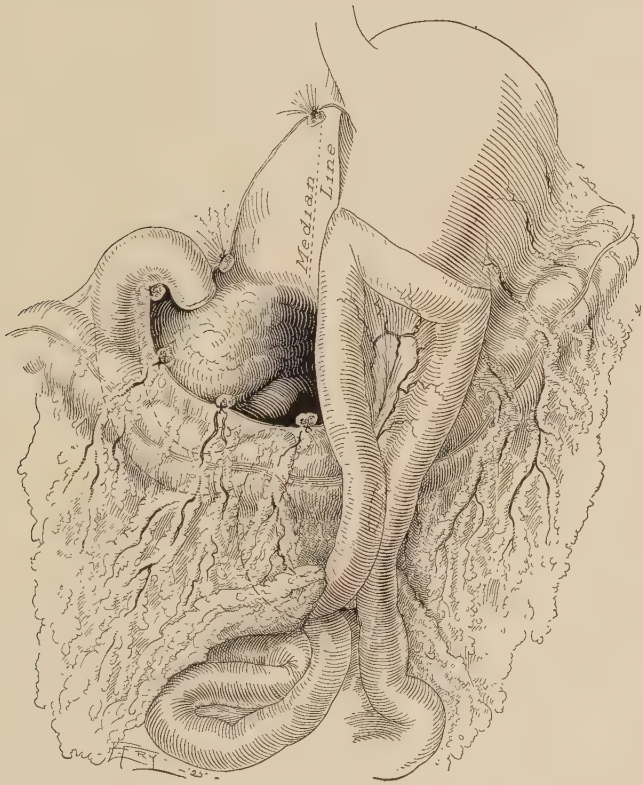


FIG. 8.—Anterior end-to-side anastomosis.

anastomosis is probably the operation of choice (Fig. 7). It is frequently found difficult, however, to arrange a posterior anastomosis properly below the mesocolic opening, and in those cases of great technical difficulty, characterized by extensive adhesions, it is safer to make an end-to-side anastomosis in front of the colon adding to this an entero-anastomosis between the loops of the

jejunum (Fig. 8). Our postoperative observations in these cases demonstrate that the results of this operation are just as satisfactory as those that follow a posterior end-to-side anastomosis, and the anterior anastomosis will at times obviate very difficult situations. It is of first importance in the postoperative care of these cases to maintain the gastro-intestinal tract as near absolute rest as possible for four or five days following operation, particular attention being directed to the control of retention by lavage.

I have recently modified the procedure just described by mobilizing the anastomosis and the jejunal loop above the opening in the mesocolon, beginning the resection at the pyloric end of the stomach before the anastomosis is disconnected and carrying the division of the gastro-colic and gastro-hepatic omenta above the anastomosis. This method provides a very excellent exposure of the entire anastomosis, and the excision of the anastomotic ring and the lesion can be carried out with very great precision.

In cases of complicated gastro-jejunal ulcer the management of gastro-jejuno-colic fistula presents the greatest difficulties, and since this complication adds materially to the risk of the operation, every precaution must be taken to carry these patients through safely. The operative procedure is the same except that the colon is first separated from the anastomosis, and the fistulous opening in the colon closed and carefully covered with surrounding omental tissues. The greatest possible care must be taken to avoid contamination and to attain secure closure of the opening in the colon. In some cases in which the fistulous opening is large or has been closed with difficulty a catheter may be advisedly sutured into the cecum.

Acute perforation of a gastro-jejunal ulcer occasionally occurs. I recently operated on a patient in whom acute perforation had taken place while he was in the hospital waiting for operation. Operation was carried out two and a half hours after the lesion had perforated, and the opening in the anterior aspect of the anastomosis was found to be about 1 cm. in diameter. There was an extensive irritative peritonitis and already much plastic lymph on the adjacent tissues. It appeared radical, under such circumstances, to carry out the operation of choice in this case, namely, partial gastrectomy, but it seemed reasonable to do so since the perforation was so recent. The anastomosis was disconnected; the indurated area, which extended well into the jejunum, excised,

and partial gastrectomy performed. The patient ultimately recovered satisfactorily.

Bleeding occurs as a complication in gastro-jejunal ulcer with greater frequency than it does in primary ulcer. It constitutes no special indication, since the removal of the ulcer is practically always a part of the operation.

Chronic perforation may occur into the abdominal wall. I recently encountered a very interesting case which illustrated the very marked tendency of gastro-jejunal ulcer to penetrate into adjacent tissues. Three operations had been performed elsewhere in 1915: anterior gastro-enterostomy was the first, but the others were not definitely determined. When the patient came to the Clinic in 1923 there was a gastro-jejunal ulcer in the anterior aspect of the anastomosis which had perforated into the abdominal wall. The ulcer was excised at this time and a plastic operation performed on the gastro-enteric anastomosis. The patient's relief from symptoms was of very short duration, and he returned in 1926 with evidences of recurring ulcer and extensive induration of the abdominal wall. Exploration revealed a perforation into the abdominal wall with a pocket 3 cm. in depth and 4 cm. in diameter. The portion of the abdominal wall, containing the base of the ulcer, was excised, the anastomosis separated, and partial gastrectomy performed. Since the last operation the symptoms have been completely relieved.

The symptomatic results following partial gastrectomy for gastro-jejunal ulcer fully justify adhering to the principle that this operation is necessary in those cases which do not permit the conservative practice of disconnecting the anastomosis only. Complete relief of symptoms follows the operation in more than 85 per cent of the cases, and the safety of the operation is shown in the mortality rate of 3.37 per cent.

I have intended to emphasize in this paper three points: (1) That the incidence of gastro-jejunal ulcer following properly performed gastro-enterostomy in well selected cases is 2 per cent; (2) that when such recurring ulceration has developed, secondary operation should be carried out without delay, and (3) that the operation of choice, provided the ulcer in the duodenum is completely healed and no obstruction has resulted, is the disconnection of the anastomosis; and if this is not advisable, partial gastrectomy promises the best prospect of cure, and any other procedures must be regarded as ineffective substitutes.

SHORT MESOCOLON AS A COMPLICATION OF GASTRIC SURGERY

By FRANK S. MATHEWS, M.D.
NEW YORK CITY

The purpose of this paper is to call attention to a short transverse mesocolon. This condition is occasionally encountered in persons in whom gastroenterostomy is indicated. It then complicates the situation, because the typical posterior no-loop method of gastroenterostomy is difficult or impossible. Surgeons must have encountered the condition from time to time, but seem not to have considered it of enough importance to call for comment. Four cases of short transverse mesocolon are here recorded which developed in the course of gastric surgery, and attention will be called to the modifications of operation which have been necessitated thereby.

Case I.—This patient was a female, with a duodenal ulcer and cholecystitis. On raising the omentum to expose the transverse mesocolon a large area of the posterior wall of the stomach was encountered instead of the expected transverse colon. An investigation then revealed that the caput coli did not descend quite to the level of the umbilicus. It was fairly free, however, and the ileum was found to enter it on its left side. The condition then was one of congenital partial rotation of the colon, the rotation on its long axis being complete, as shown by the entrance of the ileum from the left; but the descent of the caput into the right iliac region was arrested. In this case posterior gastroenterostomy was done behind the omentum, but in front of the colon. As a loop was employed, it was thought wise to add an enteroanastomosis with a Murphy button.

This case was encountered ten years ago and three others have been met in the interval up to the present time.

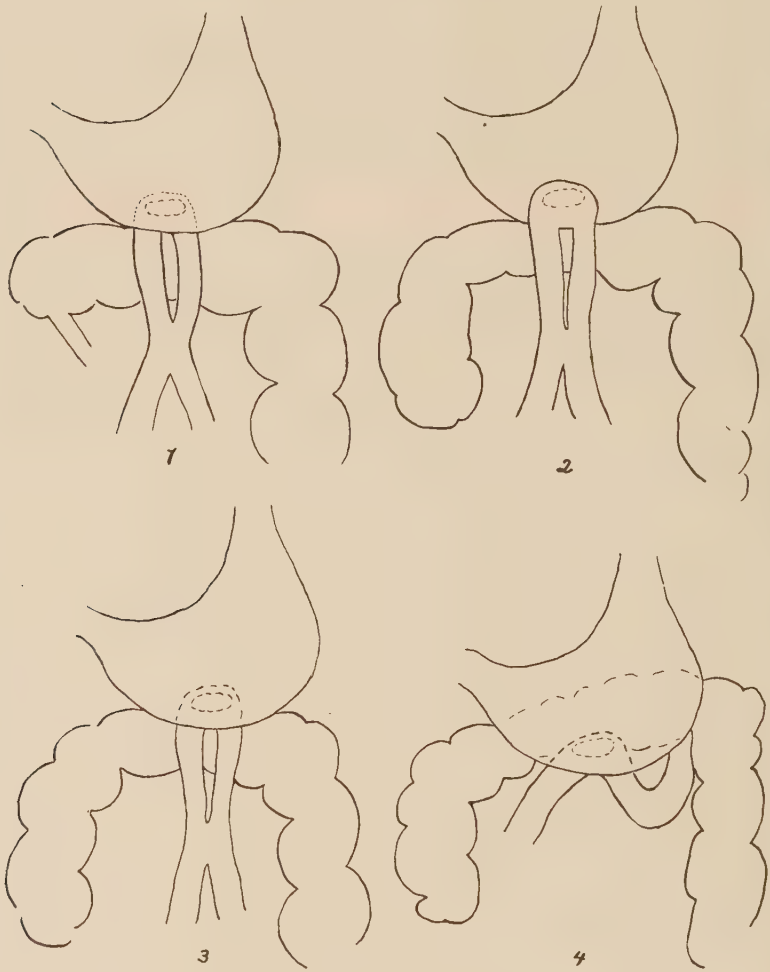
Case II.—The patient was a man, aged forty-four years, with a benign pyloric stenosis. The greater curvature of a greatly dilated stomach descended into the true pelvis. The mesocolon was found so short that a retrocolic gastroenterostomy would have been impossible. An anterior one with added enteroenterostomy was resorted to as being the best method of handling the condition. In this patient, even had the mesocolon been of the usual dimensions, a posterior no-loop gastroenterostomy would not have been a wise operative procedure because of the large size and extreme ptosis of the stomach. In such cases the no-loop operation would leave the big stomach suspended from the point of anastomosis.

Case III.—The patient, a man, aged forty years, had two ulcers—an active one on the lesser curvature some distance from the pylorus, and a partially healed, constricting, duodenal ulcer which was responsible for a considerable gastric residue. The ulcer in the stomach was excised and its bed closed by sutures. The mesocolon then being found too short to permit the posterior operation, a posterior gastroenterostomy was performed in front of the colon. A long-enough loop of jejunum was used so that there could be no possibility of its being compressed, and an enteroenterostomy was added.

In these cases, then, the procedure has been either anterior or posterior gastroenterostomy, using a considerable loop of jejunum above the anastomosis, and in each case an enteroanastomosis was added to avoid the possibility of retention of intestinal contents in the segment of bowel above the gastroenterostomy.

Case IV.—The patient was a physician, aged thirty-four year, with an active, penetrating ulcer on the anterior wall of the duodenum. The ulcer was destroyed by a cautery and the defect closed by a layer of sutures. The narrowing of the pylorus seemed to make a gastroenterostomy advisable. On delivering the omentum the whole of the back of the stomach came into view covered only by one leaf of the omentum. Investigation showed the ascending colon in normal position. The transverse colon, however, lay closely applied to the vertebral column and quite fixed. In this case an anterior colic posterior gastroenterostomy was performed, but an enteroenterostomy was not added, though the operation was not a no-loop one. The spot employed for anastomosis was about 6 inches from the duodenojejunal junction, the gut being applied obliquely to the posterior stomach wall. In this case the jejunum seemed to lie quite straight at the point of anastomosis and not to be angulated on either side of it. After operation the

patient made a conspicuously good recovery, but I was worried for a number of days thereafter because of the omission of the enteroanastomosis. After operation the x-ray plates in this case were reviewed



and found to give some evidence of the short mesocolon. The plates taken with the patient in a horizontal position, after a bismuth enema, showed the transverse colon lying rather high and straighter than one would expect. Plates taken after the bismuth meal with the patient standing showed a moderately ptosed stomach. The colon, which was

moderately outlined in gas, lay above the lesser curvature of the stomach instead of looping downward below the greater curvature of the stomach, as one might expect.

Each of these patients has done well following operation. There has been no suggestion of vicious circle in any of the 4, and the patient in whom no enteroanastomosis was made has done quite as well as the others. Nevertheless, I feel that the enteroanastomosis is an added security when a long loop has to be used.

When one encounters a case of very short transverse mesocolon it will probably not be discovered until the work on the stomach has been completed. Then, when the omentum is lifted up with the expectation of drawing the transverse colon immediately into the wound, one sees instead the large expanse of the posterior wall of the stomach covered only by a single leaf of the omentum. Posterior or anterior operation can easily be done. The posterior one would seem to be more desirable, especially if the omentum is of considerable size. But whether posterior or anterior operation is employed, a short loop of small bowel would seem undesirable because of the possibility of its being compressed by the transverse colon. The question of whether the enteroanastomosis should be added may seem a debatable one. We have always added it in carcinoma cases, where an anterior gastroenterostomy had to be made high on the stomach wall. The enteroanastomosis, whether by suture or Murphy button, adds very little to the length of operation, and may be the means of sparing the patient a second operation.

In 1 of the above cases a short transverse mesocolon was associated with a partial rotation of the colon. In the others no associated anatomic abnormalities were encountered.

DISCUSSION ON THE PAPERS OF DRS. BALFOUR AND MATHEWS

Dr. E. P. Richardson, Boston, Mass.:

I was very much interested in Dr. Mathews' paper. I have done a gastroenterostomy in 3 cases with a short mesocolon. In 2 of them it was due to a congenital anatomical condition; in the third to adhesions from operation. The anatomical arrangement was the same in all. The transverse colon was fixed very close to the spine, the stomach overlapping it in front, with the omentum hanging down from the greater curvature of the stomach. In all 3 cases it was possible to do an antecolic posterior gastroenterostomy to the posterior wall of the stomach, which hung below the transverse colon, with the line of anastomosis protected from adhesion to the anterior abdominal wall by the omentum. The function was very good in all 3 cases.

Dr. Balfour (in closing):

I would like to say a word of appreciation of what Dr. Bevan has said, because I think we ought to keep in mind all the time that duodenal ulcer is a chronic disease, and yet it is a disease which does not justify very radical treatment at any stage except in those few cases where patients fail to get well after simple measures.

One should keep in mind the sequence of management of a patient with duodenal ulcer. The first step probably would be to modify the patient's habits of living—to eat less, to rest more, to abstain from smoking, alcohol, etc.—and if such methods failed, then to go on to a more elaborate medical management. If medical treatment fails, and the disease becomes chronic, one should carry out the simplest surgical procedure which would give good prospects of satisfactory results. One of the first requisites of getting good results in duodenal ulcer is that the disease is chronic. As far as we know, the simplest surgical procedure is a gastroenterostomy, which should maintain a permanent alkalinization of gastric juices and permanently relieve intragastric tension. If this operation fails, and the patient gets a recurrence of ulceration, radical surgical measures are justifiable.

SURGICAL ASPECTS OF CERTAIN PHASES OF LIVER FUNCTION

By EDWARD P. RICHARDSON, M.D.
BOSTON, MASS.

The work of numerous investigators focussed on the liver and biliary system under normal and abnormal conditions has led to important recent advances. While our understanding of the physiology and pathology involved, our accuracy in diagnosis and effectiveness in treatment have been greatly increased as a result of these labors, the mass of new material is so great that in certain instances it is difficult as yet to choose measures of actual value in clinical surgery.

The diagnosis of disease principally confined to the gall-bladder, through clinical evidence and *x*-ray studies according to the method of Graham and Cole,[1] now compares favorably with that of any other intraäbdominal pathologic condition. The same cannot be said of surgical disease involving the liver. In fact, the more the liver is affected in surgical conditions, either directly or through disease of the gall-bladder and ducts, the less are available methods likely to give us a complete picture of the situation. Some method for the prompt separation of those cases of jaundice due to surgical conditions, some way of estimating the extent of liver damage in a given case, and, in particular, some measure of the adequacy of the liver for the biologic needs of the individual are obviously greatly to be desired.

The physiology of the liver and its disturbance in disease, therefore, have a definite bearing in surgery. The difficulty is to apply what is known of the function of the liver to the study of clinical cases. In spite of the vital importance of the liver and the multiplicity of functions assigned to it, in carbohydrate and protein metabolism, in the storage and modification of fat, in the formation of fibrinogen, in detoxication, and in the excretion of bile pigments

and bile salts, its reserve and its power of regeneration are so great that in man even up to the time of death there are few changes constantly present which we can attribute to failure of the hepatic epithelium.

Although Mann and McGath[2] have shown a progressive decrease in blood-sugar following removal of the liver in dogs leading to characteristic symptoms and death, in man a low blood-sugar during the course of hepatic disease is exceptional. Bollman, Mann and McGath[3,4] have shown in similar experiments that a profound modification of protein metabolism occurs. There is a cessation of urea production, with consequent fall of blood-urea if the kidneys are intact. Uric acid accumulates in the blood in spite of greatly increased excretion in the urine. It seems fair to assume that the liver is equally important in protein metabolism in man. Yet in disease, while findings of physiologic significance may occur in acute yellow atrophy, changes in the nitrogenous excretion in the urine or in the blood chemistry sufficiently characteristic of liver damage to be of value in routine diagnosis or prognosis, rarely occur. In fact, blood chemistry is of greater value in hepatic disease through showing the effects of nephritis or of secondary changes in metabolism than it is with reference to the liver itself.

The obscurity of changes directly dependent upon damage to the liver cells makes clinical tests of function particularly desirable. Although numerous tests have been devised, in their present stage they are chiefly applicable in the intensive study of groups of cases; few have established themselves as valuable in the clinical routine. Greene, Snell and Walters[5] give a critical consideration of the more promising of such tests. In actual practice the most helpful evidence comes from disturbance of one function, that of bile-pigment metabolism. While much valuable information may be gained by a study of the character and amount of bile secretion into the intestine, I wish to refer particularly to the retention of bile pigment in the organism. To discuss this further requires a more detailed consideration of the source of bile pigment and the relation of the liver to bile pigment formation and excretion. Since the dye-tests of liver function bear some relation to the excretion of the normal biliary pigments, it is logical to postpone reference to them until later.

The rôle of the liver in the occurrence of jaundice has been

the subject of much dispute and appears only recently to have been settled. Jaundice may be defined as the abnormal retention of bile pigment with in the body. The only known source of bile pigment is hemoglobin (Rich[6]). Conversion of hemoglobin into bile pigment occurs outside of the liver (Mann[7]), probably through the action of the cells of the reticulo-endothelial system. The introduction of a delicate test, by van den Bergh,[8] has shown that bile pigment (bilirubin) is normally present in the blood stream in a concentration of 1 to 400,000 to 1 to 1,000,000. We may look upon the epithelial cells of the liver, therefore, as excreting the bile pigment brought to it by the circulating blood, rather than producing it by their own activity. Further, the amount of hemoglobin available for conversion into bilirubin will depend on the rate of the red-cell destruction (Rous and Drury[9]), which is high in the primary anemias, and presumably greater in an individual with a normal red-cell count than with a low count due to secondary anemia. The depth of jaundice in any given case will depend on two factors, aside from irritations in pigment excretion by the kidneys, the rate of red-cell destruction and the condition of the liver.

Under this conception of jaundice the important clinical changes, therefore, are those taking place in the blood stream. The tissue-staining of gross clinical jaundice reflects only slowly and incompletely the variations of pigment concentration in the circulating blood. (Rous[10]). Since the amount of bile pigment (bilirubin) normally present in the blood plasma is considerably less than that at which clinical jaundice occurs, there may be recognizable degrees of retention short of actual jaundice. It becomes important, therefore, to measure accurately the degree of bilirubin retention, since by this means we may demonstrate latent or occult jaundice.

The actual measurement may be made by means of van den Bergh's indirect diazo reaction, which gives a violet color only in the presence of bilirubin, or by an estimation of the depth of yellow color of the serum. In the latter method the yellow color is compared with a standard solution of potassium bichromate 1 to 10,000 (Meulengracht[11]), in a Duboscq colorimeter (Maue[12]), or more simply with a series of dilutions of the same chemical in test-tubes, as suggested by Murphy,[13] the findings with regard to this arbitrary standard constituting the icterus index. Since the normal icterus index is from 2.5 to 6, there is range for an

important degree of pigment retention before clinical jaundice appears at about 15. The yellow color of the serum may be due to other substances beside bilirubin. Van den Bergh's indirect or quantitive reaction is, therefore, more reliable as well as more delicate, but requires technical assistance. We have found Murphy's[13] method, checked at times by the van den Bergh reaction, adequate for routine clinical purposes.

The estimation of bilirubin retention through the icterus index or van den Bergh test is of unquestioned value surgically, and deserves to take its place beside the guaiac test of the stools and the demonstration of red corpuscles in the urine. The finding of latent jaundice gives much the same general type of information, our attention is directed either to the biliary system and liver or to excessive red-cell destruction, which is valuable in obscure cases if too much weight is not given to it. Naturally, latent jaundice is more frequent in gall-bladder disease or septic processes in the liver than in peptic ulcer or renal colic. Further, there is now no need for argument as to whether a patient is jaundiced or sallow, it can be demonstrated beyond doubt. Finally, we have a means of measuring accurately the depth of jaundice by which we can follow the progress of disease, choose a favorable time for intervention, and estimate the effect of treatment.

The demonstration of latent or measurement of actual jaundice by these methods gives no hint as to the cause of the pigment retention. The prompt recognition of jaundice relievable by surgery is an important factor in lessening operative risk. The qualitative or direct van den Bergh test would seem to give the greatest aid in separating various types of jaundice. According to McNee,[14] jaundice may be due to excessive hemolysis, as in pernicious anemia or in familial jaundice; to toxic or infectious action on the liver epithelium, as in pneumonia and after the administration of certain drugs; or to obstruction of the bile passages.

Van den Bergh[15] has demonstrated by means of his direct diazo reaction that the retained bilirubin causing jaundice may occur in two forms: In one form it gives prompt (maximal within thirty seconds)* violet color with his reagent; in another form it gives

* In our experience according to the technic of McNee[14] the color change is rarely so prompt.

either no color or only after long delay. Experience has shown that the prompt reaction occurs typically in obstructive jaundice, and the delayed reaction in hemolytic jaundice. He explains this on the basis that in obstructive jaundice the bilirubin has been acted on and altered by the epithelial cells of the liver and is subsequently reabsorbed, while in hemolytic jaundice the excessive formation of bilirubin takes place faster than the liver cells can excrete it, hence it tends to accumulate in the blood unacted upon by the liver cells. In actual practice, types of jaundice are found which give all gradations in speed of reaction between the prompt and the delayed. McNee[14] states that these intermediate reactions, to which the name of biphasic has been given, can be accounted for on the view that both the variety of the bilirubin which gives the prompt reaction and that which gives the delayed reaction are present in the serum together. He associates these biphasic reactions particularly with the cases of toxic and infective hepatic jaundice, and explains them by assuming that some bile pigment fails to pass through the damaged liver cells and passes directly into the blood, while the remainder, which is excreted by the still active liver cells, is obstructed in the bile capillaries and is reabsorbed into the circulation.

Here is a test which at first sight should be helpful in differentiating the cases of jaundice capable of being benefited by surgery. But even if the cases of obstructive jaundice are infallibly separated by the van den Bergh reaction, a surgical lesion is not necessarily present. Obstructive jaundice, in the usual narrow surgical conception, possibly unconsciously influenced by the type of case in which surgery is useful, is due to compression or obturation of the extrahepatic ducts. But, obviously, obstructive jaundice can equally well be caused by obstruction of the biliary passages within the liver, due to inflammation, fibrosis or metastatic malignancy.

The demonstration of obstructive jaundice, therefore, is not enough to bring cases within the surgical group. Should the finding of a delayed type of reaction influence us in advising operation? McNee[16] states that the qualitative reaction may enable a positive diagnosis of obstructive or hemolytic icterus to be made, and that as a rule in the commonest type of jaundice, the toxic and infective group, no information of diagnostic or prognostic value is given to the clinician. Andrewes[17] concludes

that frankly obstructive can be distinguished from frankly hemolytic jaundice by means of the test, but as a means of differentiating icterus from liver-cell damage from other types of icterus the value of the method is slight. Our own observations, for which I am chiefly indebted to Dr. G. S. Speare, have shown not only that both the prompt and the delayed types of reaction may take place in catarrhal jaundice and in cirrhosis, but that the delayed type of reaction may occur in cholecystitis without duct obstruction, and in a few cases with a low serum bilirubin apparently obstructive in character, as shown by operation. Therefore, although the qualitative van den Bergh reaction is of value in clinical and experimental study of disease, as evidence for or against operation in the individual case it is distinctly subordinate to the clinical picture.

Since the demonstration by Abel and Rowntree[18] that phenoltetrachlorphthalein was excreted by the liver in the bile, much attention has been given to this dye test of liver function. When Rosenthal[19] changed the criterion of liver efficiency in dealing with the dye after intravenous infection, from the excretion in the bile to retention in the blood stream, the test became relatively simple and direct. In actual clinical use the method has certain limitations. While experimentally, as Rosenthal[20] has shown, dye retention is proportional to the amount of liver excised, in disease only one phase of liver function is tested; no information is given, except by inference, as to the state of other liver functions. If the liver is unable to excrete bilirubin normally, so that it is retained by the serum, dye retention also with few exceptions occurs. While the degree of dye retention does not parallel absolutely the depth of jaundice, there is a general relation between the two. It is not yet apparent that in cases of obstructive jaundice greater significance can be given, in diagnosis or prognosis, to the degree of dye retention than to the amount of bilirubin retention as shown by the icterus index. Further, the use of phenoltetrachlorphthalein in the doses usually recommended, 5 mg. to the kilo, is not without possibilities of harm. Thrombosis of the injected vein not infrequently occurs and general reactions have been reported. Maurer and Gatewood[21] and Rosenau[22] have called attention to the possibility of injury to the severely damaged liver. In a case shortly to be operated on this aspect is important.

The chief value of the method lies in the fact that dye retention may occur in cases where the serum bilirubin is within normal limits. Greene, McVicar and Rowntree[23] have shown that this retention may occasionally furnish the only evidence of metastatic nodules in the liver. During recovery from catarrhal jaundice and after the relief of obstructive jaundice, dye retention may persist longer than elevation of the serum bilirubin. Shattuck, Brown and Preston[24] state that the Rowntree-Rosenthal test seems to be of greater value than the icterus index in the diagnosis of cirrhosis and of malignant metastasis of the liver.

Since the introduction by Rosenthal[25] of a new dye, bromosulphalein, by means of which a test can be secured with a smaller dosage (2 mg. to the kilo), we have made use of it in a small number of cases. While it is too early to speak of results, we are encouraged to continue, both from the information secured in occasional borderline cases of liver disease and from the freedom from sequelæ.

The jaundiced surgical patient presents a complicated situation, in which disturbance of the kidneys and pancreas, dehydration, malnutrition and sepsis may share as well as interference with the liver. While the information secured by the methods discussed is of much value, it does not constitute a short cut by which a diagnosis or an evaluation of the risks of operation may be arrived at. Knowledge of the condition of the patient gained through a thorough clinical study, including the chemistry of the blood and urine, must be considered of greater importance than direct tests of liver function. The most conclusive evidence of an obstruction of the extra hepatic ducts is a persistent absence of bile pigment from the intestinal tract. In prognosis, whether or not we may refer it to liver function, the most valuable sign is a delay in the coagulation time of the blood. But in tendency to hemorrhage, as in judging the state of the liver, we should be guided by suspicion rather than by actual demonstration. If such suspicion exists, the preoperative measures emphasized by Walters[26] should be employed, an abundant fluid and carbohydrate intake, given if necessary by rectum or by hypodermoclysis, and the intravenous use of calcium chloride.

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THE GALL-BLADDER OF 1926

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The gall-bladder is still elusive as to some of its functions, and what has been learned concerning it, as well as the function of the biliary apparatus, continues to maintain our interest in it, if anything to a higher tension than ever. Abdominal surgery largely developed from 1890 to 1900, during the ten years following the decade of the discussion of the germ theory of disease and the practice of antiseptic and development of aseptic surgery: a period in which surgeons were constantly brushing up their knowledge of the anatomy of the gall-bladder, ducts and adjacent structures preparatory to operations on them. Without enlargement of the lymph nodes of the ducts lesser degrees of gall-bladder disease could not be diagnosed or even recognized with any degree of certainty with the abdomen open, and most operations on it were performed following several attacks of colic. It was often necessary for the yellow flag of jaundice to be spread before adequate effort would be made to relieve the disease. Many laymen and even some medical men considered the attacks of colic neuralgia of the stomach unless the victim became jaundiced; to refute the suggestion of probable gall-stones they pointed to the absence of jaundice. The gall-stone was the essential factor, the real disease which in some unknown and mysterious manner had developed in the gall-bladder. Catarrhal jaundice was recognized as an infection of the biliary ducts, a disease occurring sporadically or occasionally, several cases in a community. Less was known of those not rare cases of jaundice showing epigastric soreness and pain in the back, with gastric symptoms, but without colic, chill or fever, in which disease of the head of the pancreas (either interstitial or hemorrhagic) causes obstruction of the duct. Surgeons saw obstruction of the common duct in all degrees of

severity and in all stages of development following recurring attacks of colic due to Fenger's floating ball-valve stones. Blockage of the cystic duct and secondary infection caused empyema and thickening and contraction of the gall-bladder wall which forced stones into the common duct. This condition closely followed Courvoisier's observation that in the cases of stone in the common duct the gall-bladder was found contracted in 84 per cent of cases. Jaundice due to obstruction of the lower end of the common duct, from other causes than gall-stones, usually dilated the gall-bladder. It was caused by cancer of the pancreas, stricture of the common duct, or tumor of the ampulla of Vater. Pathologists now reported added dangers from a fixed stone causing no pain, as usually only the moving stones cause colic. Several years after the cessation of such attacks of colic, painless jaundice might result from primary cancer of the gall-bladder and ducts spreading to the liver, but in association with gall-stones in nearly all cases. Preservation of the gall-bladder was the general surgical rule even when it was incised for stones fixed in the cystic duct, and much stress was laid on various suitable methods of opening the gall-bladder for drainage. A few surgeons developed the operation of cholecystotomy, removing stones and immediately closing the gall-bladder by suturing. Operations for relief of symptoms of gall-stones, as diagnosed, showed that there were other diseases of the gall-bladder besides gall-stones, such as the so-called "acute abdomen," an inflammatory condition often associated with interacinar hemorrhagic pancreatitis and fat necrosis, commonly seen in obese patients. Catarrhal cholecystitis with thickening of the mucous membrane sometimes showing yellow lipoidal degenerated spots in the thickened dark-red mucosa (and from its appearance designated "strawberry gall-bladder"), or large and small mucous papillomas were recognized. Past inflammation of the serosa was indicated by adhesions to the duodenum, colon, or omentum. Cholecystostomy with drainage for the latter condition frequently left the patient worse than before operation from the adhesions of the fundus to the abdominal wall and the traction colic they caused. In the meantime patients who had had gall-stones removed years before by cholecystostomy returned with former symptoms and the gall-bladder was again found filled with stones. In some cases three operations for stones were required, or a second operation for nonfunctioning

gall-bladder, seen so frequently as a result of disease. Thus from 1910 to 1915 cholecystectomy was developed as the operation of choice. It was now generally appreciated that the gall-stone was the result of a condition in which the gall-bladder played an important part, and more and more investigative research was directed to the biliary apparatus. A knowledge of the causation of gall-stones and inflammation of the gall-bladder is said to be necessary for proper treatment. I do not agree with this as we have made all our progress in the medical and surgical treatment of gall-stones and gall-bladder disease without knowing the answer. The surgical treatment is satisfactory. Knowledge of the cause of the disease is essential for prevention of it and to make possible better care following operation; and all surgeons are interested in it.

The gall-bladder is present in most animals which secure their food from the surface of the ground. The leaf-eaters which are mostly antler-bearing and cast their antlers yearly have no gall-bladder. Other leaf-eaters like the elephant and horse also have no gall-bladder. The pocket gopher, living continually beneath the surface, has no gall-bladder, while the striped gopher, living beneath and feeding above, has one.

Embryologically the gall-bladder begins in a small nest of cells from which are developed the pancreas, liver, duodenum, and ducts. It occurs as a solid bud on the common duct which later becomes hollow and enlarges, and consists of four coats, mucous membrane, submucous layer in which are large and active lymphatics, muscularis with elastic and connective tissue, and serosa, beneath which is a poor arrangement of lymphatic vessels. It is supplied by a large artery, the cystic, and a few vessels join it from its attachment to the liver. The gall-bladder receives circulation with full heart pressure while the liver is supplied by a comparatively small artery to cells all alike working under low tension. The gall-bladder in man holds approximately 30 cc. of bile, probably one-fortieth or less of the total amount of bile made in twenty-four hours. Early morning operations on the abdomen usually show the gall-bladder well filled (the fasting condition). The liver apparently makes bile at varying speed but constantly, and varying amounts pass into the duodenum according to the digestive activity. It is most active following meals until the food has left the stomach. Mann believes that

from this time until the next meal the gall-bladder probably deals with most of the bile by filtration of it through its wall into the lymphatic tract. With his associates he has shown that tension is not the same in the gall-bladder and in the duct, indicating that the gall-bladder can contract and close the cystic duct at its juncture with the pelvis of the gall-bladder. The cystic duct is surrounded by muscle and usually has folds of mucous membrane which are alternately or spirally placed about the duct and were described by Heister, in 1782, as valves; yet some cystic ducts have no valves whatever. The cystic unites with the common duct at an acute angle or the hepatic duct is separated from the opening of the cystic duct by an intervening septum extending below the cystic. The gall-bladder not being like a rubber bulb can create no vacuum for suction. It is dependent then on the closure of the outlet of the common duct into the duodenum for bile to fill the viscus, through the cystic duct. Gage, in his examination of the common duct of the cat in 1879, showed that a sphincter existed at the outlet. Oddi made extensive research on many species of animals, and proved the presence of this sphincter. It is also found in types of animals which have no gall-bladder; having no gall-bladder they have proportionately slightly larger ducts.

It has been shown that the bile in the gall-bladder of man is approximately ten times as concentrated as it is possible for the liver to make it or as found in the hepatic ducts. Bile is rather essential for the digestion of fat, yet there is marked variation in the amount of fat in the natural foods in different types of animals which have gall-bladders. It is probable that the gall-bladder has control of its own cystic duct in order to cause pressure-filtration of the water or fluids of bile which pass to the blood stream through the lymphatics without coming in contact with the intestinal contents; also to work properly it should have control of the sphincter of Oddi in order to refill itself. Sweet and numerous others believe that under normal conditions little or none of the bile which enters the gall-bladder leaves it by way of the duct. He has demonstrated parietal pockets along the larger intrinsic and extrinsic hepatic ducts which might also act as filters if we can think of them as serving this purpose. The so-called glands of Luschka, little pockets in the mucous membrane of the gall-bladder, are undoubtedly not true glands. These correlated

actions are probably somewhat like the law of contrary innervation propounded by Meltzer and act through the nervous system, the sympathetic and parasympathetic. Whatever amount of bile is found in the gall-bladder should then represent ten times as much bile as has entered it. The flow of bile into the duodenum is said to be increased by intraäbdominal pressure which occurs by respiratory efforts. This probably has some effect but not in fish because they respire through gills; the principle must serve varying conditions. Coffey showed that the common duct traverses the wall of the duodenum for from $\frac{3}{4}$ to $1\frac{1}{4}$ inches. The rolling peristaltic waves during digestion would constantly milk the common duct and aid in delivery of bile when most needed. It is difficult to estimate the amount of exudate of mucus as a colloid formed by the mucous lining of the gall-bladder. After cholecystostomy, with a blocked cystic duct for any reason causing fistula, a considerable amount of clear mucus is discharged daily. Whether this mucus has the power of solvent action on inspissated bile has not been sufficiently studied. Aschoff showed that healthy bile itself has some power of absorption of gall-stones and that small stones placed in a healthy gall-bladder lose a considerable part of their weight during a few weeks. Blocking the lower end of the common duct without gall-stones will distend the gall-bladder and ducts. Within a few weeks, first dark bile, then inspissated bile, granular and thick, fills the gall-bladder and ducts, and in a few more weeks white bile as colloidal mucus alone is found filling the gall-bladder and ducts. Mann and Bollman, by ligating the common duct in animals, have shown that a noticeable increase in bilirubin is not found in the blood sooner than from twenty-four to thirty-six hours and jaundice does not show for approximately four days, indicating that the gall-bladder can filter a very large amount of bile. If the ligation is made and at the same time the gall-bladder removed the same increased amount of bile salts is noticed in the blood in from three to six hours and jaundice appears in twenty-four hours. Sheard with spectrophotometric studies shows that it is increased in each fifteen minutes.

Nature has placed on each of the common, hepatic and cystic ducts at least one lymph node and occasionally two. They have a definite size in health, and all surgeons who have paid attention to them have depended quite largely on evidence fur-

nished by their enlargement for indications of an excess of filtration by the gall-bladder. The size of these lymph nodes always depends on the degree of overwork or the degree of infection present.

For a few decades we have had surgical treatment of gall-stones and gall-bladder disease and previous to that the medical treatment; now medical drainage of the gall-bladder and ducts has been added. It is accomplished by passing a tube through the stomach into the duodenum and inserting a solution of magnesium sulphate or of oleic acid. This relaxes the sphincter and stimulates the flow of bile; gall-bladder bile evidently constitutes a part of this quantity of bile. Food passing from the stomach as chyme also stimulates the flow of bile with relaxing of the sphincter, while a 3 per cent solution of hydrochloric acid applied to the duct area raises the duct pressure to over 800 mm. through spasm of the sphincter. There is very little (from 10 to 30 mm.) back pressure in the common and hepatic ducts of animals without a natural gall-bladder, and the same condition obtains after removal of the gall-bladder. The tension in the common duct taken under anesthetic runs 75 to 100 mm. of bile; that in the gall-bladder varies from 100 to 200 mm. Therefore such a pressure is capable of filtering the fluids from bile and of increasing its salts to ten times its manufactured strength. Rough handling and crushing of the wall of the gall-bladder in forceps seem to check its activity of filtration. Thus fresh bile with dye (Graham-Cole test) does not enter the gall-bladder to show in the roentgenogram. Disease of the gall-bladder also checks its activity of filtration. Mann was able to show that chemical inflammation of the gall-bladder produced by injecting Dakin's chlorine solution intravenously, which has a selective affinity for the gall-bladder, greatly checks its activity of absorption, depending on the degree of injury produced. The liver has selective affinity for certain dyes, as was noted by Rowntree in 1914 in working on the chemical tests of renal and hepatic function. He showed that phenolsulphonephthalein was excreted by both the kidneys and liver and that a chlorine derivative, especially phenoltetrachlorphthalein, was handled solely and specifically, by the liver. This is the basis of the Rowntree-Rosenthal test of hepatic function. Mann has shown that when Rose Bengal dye is given intravenously 50 per cent will appear in the gall-bladder within one hour; some of this

appears in the gall-bladder when the cystic duct is ligated, indicating that it can pass into the gall-bladder from the vascular system either from the liver or the cystic artery.

The Graham-Cole test is commonly used to demonstrate gall-bladder disease with dyes excreted by the liver. A normal gall-bladder takes the dye-stained bile into itself through the cystic duct, and shows in the roentgenogram. If there is any interference with the passage of bile into the gall-bladder its outline shows but faintly or does not show at all in the roentgenogram. Single cholesterin stones may appear in a gall-bladder so little diseased, that it will show the dye. The modern method of giving these dyes by mouth is sufficiently effective and obviates the reaction often noted when they are given intravenously or by hypodermic. This test, however, should more frequently be reserved for the doubtful cases, difficult of diagnosis. Inflammatory conditions of the gall-bladder are infective and due to the toxins produced by the various bacteria.

Three routes of entrance of infection have been discussed: (1) Bacteria from the intestine enter the common duct and pass into the gall-bladder through the cystic duct; (2) they pass through the liver through the portal circulation and are not completely destroyed by the liver and pass into the gall-bladder with the bile; (3) they are backed up through the lymphatic system of the gall-bladder, thus gaining entrance through its wall. These are all possible but not probable causes. Pure bile is not a good culture medium for types of bacteria found in gall-bladder disease. When inflammation is present and the bile contains 30 per cent of serous exudate, it is a fairly good culture medium. This presupposes an inflammatory condition making the gall-bladder and its contained bile vulnerable to the attack of bacteria. Infected material from the wall of an acutely inflamed gall-bladder carefully prepared by Rosenow to remove superficial contamination will give a culture of these bacteria. When they are intravenously injected into animals acute inflammatory disease of the gall-bladder results in over 70 per cent of tests. These animals being healthy and not ready of themselves to develop gall-bladder disease soon recover from the local disease. The hematogenous route is undoubtedly the common one. The gall-bladder, once infected, becomes a probable focus for aggravation of existing disease of the heart muscle.

The normal liver is a peculiar dark red with a fairly sharp or axe-like edge. The abnormal liver found associated with gall-bladder disease and probably preceding the gall-bladder disease is darker, roughly mottled, often adherent to the parietal peritoneum or omentum from previous attacks of hepatitis; from its color, and rough and granular appearance it could be said to be in a state of biliary congestion or retention If the sympathetic system now produced increased tension on the common duct through spasm of the sphincter of Oddi doubling its pressure, the gall-bladder could be made to overwork and filter an excess of bile fluids, leaving the residue considerably more concentrated than normal, in fact tangible and visible as bile sand, consisting of bilirubinate of calcium, bilirubin and bile salts with cholesterin. With but little increase in tension the duct may not relax during digestion and thus greatly increase the work of the gall-bladder and temporarily cause gray or light-colored stools. In some quick and fleeting attacks as seen in pregnancy, almost clear cholesterin constitutes the stone with but little change in the liver or gall-bladder. Gall-stones undoubtedly form quickly, even in a few hours through the stimulation of excessive fat or from toxins. Usually a new stone is formed, but occasionally an extra layer is deposited on a previously formed stone, the total material usually being less than that daily handled by a hen in forming the egg-shell. Thus stones are almost never found in the process of formation but they vary in density with their age in the same gall-bladder. Are the bacteria the cause of the condition, or do they reside in the gall-bladder wall because of some liver disease and become active under opportunity? Sometimes clumps of bacteria are found in the center of gall-stones, but Rosenow's idea of selective affinity of bacteria for the gall-bladder wall through the circulation is undoubted the correct one as its interior should be as well protected as the hepatic and cystic ducts. With bacteria alone Rosenow has created minute gall-stones in the natural pockets in the wall of the gall-bladder of animals by intravenous injection of bacteria from diseased gall-bladders. A congested liver with diseased gall-bladder usually shows, above and about the attachment of the gall-bladder, whitened areas of connective tissue. This area is rich in lymphatics which are associated with those of the gall-bladder. It is a part of the filtration area used by the gall-bladder. This area is thrown into relief immediately

after an intravenous injection of Dakin's solution of chlorine into animals, showing chemical inflammation to be a counterpart of bacterial inflammation.

The study now becomes more interesting. A diseased gall-bladder overworked in filtration and with a bile-congested liver brought about by spasm, elevating the tension but not blocking the sphincter of Oddi, should accumulate residue; this should precede the development of gall-stones. We believe there is an increase in the percentage of cases of diseased gall-bladder, and appendix, and of gastric and duodenal ulcer, most of them not recognized until gross lesions have occurred. Appendicitis, formerly worthy of extensive discussion, is now accepted as a disease entity and operations on it are common. The more seriously it is found to be affected with chronic disease in middle age, the more probably will it be accompanied by disease of the gall-bladder or a duodenal or gastric ulcer.

The McBurney incision for appendicitis should only be made in children under sixteen years of age; over that age abdominal investigation is best made through a straight incision in the rectus and an infected appendix is best dealt with through a similar incision. There is undoubtedly an etiologic relationship between appendicitis and disease of the gall-bladder in middle life. There are few necropsies of persons aged forty years or over in which the appendix is normal. This is also the case today with the liver and gall-bladder. Spasm of circular muscle occurs through the mild stimulation of the sympathetic, but may also be apparently of vascular origin, attributable again to the sympathetic.

Is it not possible that, varying with the individual, some defect or deficiency in food intake, still remaining to be discovered, may cause a spasm of the sphincter of Oddi, doubling the work of the gall-bladder filtration, often causing secondary spasm of the pylorus, or plaque spasm of areas of the stomach wall, with or without the association of appreciable gall-bladder disease, or that spasm of the circular muscle bands, interfering with the circulation of local areas of the mucous membrane of the appendix or the colon in diverticulitis, makes such areas less resistant? The variation in activity, degree of virulence of the bacteria or lowered resistance would explain the difference in the acute or chronic character of the disease or the amount of destruction. Such reasoning would assign to bacteria the rôle of either primary

or secondary agents of disease not necessarily in the gall-bladder wall but primarily in the intestine, frequently with additional foci in the mouth, the cervix uteri after the age of forty, and the prostate, after the age of forty-five years. It may be that we have overlooked the sympathetic nervous system as an associated agent of disease when locally influenced by one or more of several possible causes.

Is it not possible that spasm of circular muscle, originating in the sympathetic nervous system is the primary basis of many diseases of the appendix, of diverticulitis, of gall-bladder disease analogous to Raynaud's disease of peripheral vessels, or sudden spasm of the renal circulation? May not the sequence of gall-bladder disease be: (1) Spasm of the sphincter of Oddi, due to some unknown cause, perhaps by food deficiency or toxins; (2) overwork in the function of filtration; (3) inspissation of bile; (4) altered nutrition of the gall-bladder tissues due to circulatory changes which are secondary to spasm; (5) bacterial changes, the type of disease varying with the virulence of the organism, the nature of the local condition, duration and like conditions? In other words, may not sympathetic irritation and muscular spasm be at times a primary clinical factor worthy of further investigation?

Urticaria has long been considered as caused by some particular type of food which is toxic for the individual; others develop eczema or nasal irritation. Such individuals are fortunate as they are led to investigate and eliminate the cause. Similar manifestations of foods which are toxic to the individual unfortunately may not be evident externally. They cause diverticulitis of the colon and irritation of the sympathetic system, producing spasms of circular muscle, and producing appendicitis and gall-bladder disease secondary to long-continued biliary congestion. The vascular spasm seen in renal spasm and in Raynaud's disease serves as a link to attribute similar causes to these diseases and duodenal and gastric ulcer.

ASSOCIATION OF CHOLECYSTITIS WITH DUODENAL ULCER

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In the last 100 operations for gall-bladder disease I have met with 5 cases in which duodenal ulcer was also present, although unsuspected either from the clinical examination or with the roentgen-ray and 1 case in which the two conditions were recognized as coexisting prior to operation. Although only a few isolated references can be found in the literature, most surgeons must have had a similar experience. Infection may reach the gall-bladder by direct extension through the duodenal papilla or by the lymphatics, in which case an ulcer of the duodenum is a possible starting-point; or through the arterial circulation. It has been established that certain pathogenic organisms show a special affinity for some particular organ and tissue, and it is now believed that the blood stream is the principal route through which the gall-bladder is infected. Rosenow has shown that streptococci and colon bacilli from acute cholecystitis, intravenously injected, give rise to a like disease in animals. Is it not possible that these or other organisms may also cause ulceration of the duodenum? Healed duodenal ulcers have been found post-mortem in patients who gave no past history of digestive disturbance. In *Surgery, Gynecology and Obstetrics* (January, 1924), the late Dr. Archibald MacLaren reports 5 cases in which there was the coexistence of cholecystitis and duodenal ulcer. The late Dr. A. J. Ochsner stated in a clinical lecture that he had observed the two conditions associated in the same patient on several occasions.

It is clear that we can no longer rely upon occult blood as indicating a gastrointestinal lesion, as this may occur in infections of the gall-bladder. Dr. E. S. Judd, in a paper read before the Southern Surgical Association in December, 1921, entitled,

"Bleeding Ulcer of the Duodenum Associated with Cholecystitis," reports 4 patients upon whom he had operated for bleeding duodenal ulcers, and in whom the pathologic condition was more extensive in the gall-bladder than in the duodenum. In each case there was a very severe grade of cholecystitis with gall stones. The chief symptom in each case was gastrointestinal hemorrhage, usually very severe and occurring at intervals of a few months. All 4 patients complained of mild dyspepsia, but none of them had severe pain and it was impossible to elicit a history of gall-stone colic or any other symptom suggestive of disease of the gall-bladder. In 3 cases there were small areas of duodenitis in the region of the duodenal cap, with extensive hepatitis and cholecystitis with stones and infected bile. In 1 case hemorrhage occurred regularly every few days. At operation he was not able to find an ulcer, but found cholecystitis with stones and extensive scarring and edema of the liver, which oozed a great deal as the gall-bladder was removed. Several years ago Crispin, in the Mayo Clinic, studied a series of cases of gall-bladder disease and found a history of gastrointestinal bleeding in about 5 per cent. He says that in nearly every instance the bleeding ceased after the removal of the gall-bladder. Deaver reports profuse bleeding in hemorrhagic infections of the biliary tract. In 1 of his cases blood reached the duodenum through the common duct and then regurgitated to the stomach, the primary cause being streptococcic cholecystitis. Kelling, in an article on the relation between cholelithiasis and ulcer of the duodenum, in speaking of the differential diagnosis between gall-bladder disease and ulcer of the duodenum, says that occult blood with duodenal ulcer may mean nothing; it may also come from infections in the gall-bladder. Mix reports 2 cases of combined ulcer and cholecystitis, clinically diagnosed as such and confirmed at operation. The chief symptom in both cases was epigastric pain in 1 case, coming on after food; in the other, having no relation to eating. He based his reason for diagnosing gall-bladder trouble in both cases on foul breath, headaches, vomiting of bile and the sensation of upward pressure experienced by both patients. There had been no attacks of biliary colic or jaundice. As Mix points out, the cardiac symptomatology is rare in ulcer and very common in gall-bladder disease; but the presence of a good deal of bile in the vomitus I do not think is uncommon in ulcer.

CASE I.—M. S., a female, aged fifty-seven years.

Chief Complaint. Epigastric pain coming on about two hours after food. She had three attacks of severe pain in the right hypochondriac region which necessitated hypodermics of morphine. The last attack of this nature was followed by intense jaundice which lasted sixteen days. She had lost 30 pounds in weight in the last year and lived in dread of taking food.

Past History. Severe pelvic infection at the birth of her last child, eighteen years ago.

Physical Examination. The patient is well developed but poorly nourished. The lower ribs were prominent, the skin very dry and the sclera jaundiced. Roentgen-ray examinations on two separate occasions showed pressure on the duodenum between the second and third portions; the caput regular in outline. In spite of a negative roentgen-ray examination for ulcer of the duodenum a preoperative diagnosis of ulcer was made, accompanied by cholecystitis with stones.

Operation. The stomach was found to be small and thick-walled. There was an ulcer on the anterior surface of the duodenum $\frac{1}{2}$ inch from the pylorus, $\frac{3}{4}$ inch in diameter and containing new-formed bloodvessels. Palpation revealed no stones in the gall-bladder, cystic or common duct. The gall-bladder was opened at the fundus, when the mucous membrane was found to be red, thickened and four small stones were removed. On account of the patient's poor condition, cholecystostomy only was done and the duodenal condition left for future operation if the symptoms necessitated it. The patient made a good recovery, and is entirely relieved of the hunger pain and dread of eating, now three years since operation.

CASE II.—A. R., a female, aged sixty-two years. Healed duodenal ulcer; eight small faceted gall stones; chronic appendicitis. The patient gave a history of stomach trouble and nervousness for a number of years. Two and a half years previous to my seeing her she was examined by a consulting physician, when the roentgen-ray report was negative for duodenal ulcer and gall stones; the gastric analysis was also negative. At this time she complained of epigastric pain, and was tender over the transverse colon. A diagnosis of colitis and psychoneurosis was made. Just before I saw her another roentgen-ray examination was made with a negative result for ulcer of the duodenum, but evidence of external pressure on the stomach from behind. She now complained of pain in the epigastrium and gas in the stomach, and my diagnosis was gall stones.

At the operation the scar of an old healed ulcer was found on the anterior surface of the duodenum $\frac{1}{2}$ inch beyond the pylorus, with an

adhesion between the duodenum and the transverse colon and the edge of the liver just external to the gall-bladder. Eight stones were removed from the gall-bladder and one from the common duct. The appendix was thickened and obstructed and was removed.

The patient made a good recovery. I have recently heard from her (two years after the operation), and she reports that she is enjoying good health, with an absence of all her former symptoms.

Case III.—M. A. P., a female, aged sixty years. Obstructing duodenal ulcer with gall stones. For several years the patient has had pain in the epigastrium, with periodical attacks of vomiting of large quantities of fluid and partially digested food, and has steadily lost weight. Two years previous to my seeing her a roentgen-ray examination was made with a diagnosis of stenosing duodenal ulcer and no gall stones seen. Following the roentgen-ray examination the patient was much improved, had less pain, less vomiting and gained in weight, over a period of one year and eight months. For the last four months she has had a recurrence of symptoms and was referred to me for operation.

Diagnosis. Obstructing duodenal ulcer.

Operation. An indurated ulcer was found on the anterior-superior surface of the duodenum, 3 by 2 cc., with scarring and puckering. The stomach wall was two or three times its normal thickness and dilated to twice its normal size. A posterior gastroenterostomy was done. The gall-bladder was found filled with multiple small stones, which were removed.

The operation resulted in complete relief of the patient's symptoms, and she recently reported, three and a half years since operation, that she has had no recurrence and has recovered her normal weight.

Case IV.—E. B., one female, aged fifty-two years. Cholecystitis, with one large gall stone; duodenal ulcer; appendicitis. The patient had been aling for several years. Four years ago she was seen by a consultant, who diagnosed cholecystitis, chronic appendicitis and oral sepsis. She now complains of recurrence of her former symptoms, viz.: pain in the epigastrium, occurring at irregular intervals, nausea and belching of gas; no hunger pain and pain not relieved by eating. When I examined her there were tenderness and increased resistance over the appendix and gall-bladder regions. The roentgen-ray examination was negative for duodenal ulcer.

Diagnosis. Cholecystitis and chronic appendicitis.

Operation. The gall-bladder was found to be considerably thickened and contained one large stone. The appendix was chronically inflamed. A very large healed ulcer on the anterior-inferior aspect of the first

portion of the duodenum was found, but causing no stenosis. The gall-bladder and appendix were removed. As she had not had the usual symptoms of duodenal ulcer, and as the ulcer appeared to be healed and as her condition was not very good, it was thought best not to operate upon the stomach. It is now six months since the operation, and last week her husband (a practising physician) informed me that she had complete relief of her symptoms, had gained in weight and was feeling better than she had felt for years.

Case V.—W. H., a male, aged sixty-five years. This patient had indigestion and attacks of pain in the epigastrium for ten years. Two years previous to my seeing him he had a gall-bladder operation by another surgeon. A few months after this operation his symptoms recurred, with pain one or two hours after eating. For the last two or three months he has had periodical attacks of pain with vomiting of large quantities of partially digested food. He has had gastric lavage twice a week for two months, when retention, of 1 to 2 quarts, was found.

Examination. Patient is greatly emaciated. The stomach is dilated, occupying about one-half the abdomen. The passage of a stomach tube revealed about 1½ pints of retention. Patient was given glucose interstitially and a stomach lavage every six hours for two or three days to prepare him for operation.

Diagnosis. Duodenal ulcer, with stenosis.

Operation. At operation I found a huge duodenal ulcer on the anterior-superior surface of the first part of the duodenum just external to the pylorus, causing almost complete obstruction. The stomach was greatly dilated and thickened. Considering the evidence of long-standing disease in the duodenum, it seems more than probable that the ulcer was present at the time of the former operation but was overlooked. A posterior gastroenterostomy was done.

He made a good recovery, and now, six months after operation, has gained 20 pounds in weight.

Case VI.—E. A. H., a male, aged forty-eight years. When the patient entered the hospital he had been ailing for three months, and for the past three weeks had had marked jaundice, dark urine, with recurring chills and an evening rise in temperature to 103° to 104° F., and pain in the hypochondriac region. On examination, he was found to be very tender over the gall-bladder area and along the lower border of the liver, which extended about 2 inches below the costal margin. He gave a history of having had pain three or four hours after meals (relieved by taking food) and tarry stools for the past ten years. Six

Case.	Sex.	Age.	Duration of symptoms, years.	Symptoms.	Diagnosis.	Operation.	Result.
M. S.	F.	57	3	Biliary colic with jaundice; pain 2 or 3 hrs. after meals; roentgen-ray positive for all stones; negative for duodenal ulcer	Duodenal ulcer with gall stones	Gall stones with small hard pancreas and healed duodenal ulcer; obstructed appendix; patient's condition not good enough to deal with duodenal ulcer; cholecystostomy; appendectomy	Relieved.
A. R.	F.	62	3	Nervousness and epigastric tendernes; roentgen-ray negative for gall stones and duodenal ulcer	Gall stones	Several small stones removed from gall bladder; scar of an old healed duodenal ulcer; appendix thickened, obstructed and removed; cholecystostomy and appendectomy	Relieved.
M. A. P.	F.	60	2½	Pain in epigastrium; periodical attacks of vomiting; roentgen-ray diagnosis, chronic stenosing duodenal ulcer; no gall stones	Duodenal ulcer	Duodenal ulcer; multiple small gall stones; gastroenterostomy and the removal of gall stones.	Cured.
E. B.	F.	52	4	Tenderness over gall-bladder with biliary colic	Cholecystitis with gall stones	Gall stones and large duodenal ulcer; chronic appendicitis; cholecystectomy; appendectomy	Relieved.
W. H.	M.	65	5	Indigestion; pain after eating; vomiting due to retention	Cholecystitis; duodenal ulcer with stenosis	Gastroenterostomy	Cured.
E. A. H.	M.	48	10	Pain 3 or 4 hrs. after eating; tarry stools; last 4 mos., tender gall-bladder; 3 wks. jaundice	Duodenal ulcer; cholecystitis; cholangitis	Enlarged liver; thickened gall-bladder; no stones; head of pancreas hard; healed duodenal ulcer; cholecystostomy	Died 3 mos. later from cholangitis.

years ago he had what was called an exploratory laparotomy done, when the gall-bladder and duodenum were examined and found normal, but a chronic appendix was removed.

Diagnosis. Cholecystitis and cholangitis.

Operation. The liver was found considerably enlarged, extending 2 inches below the costal margin. The gall-bladder was greatly thickened, as also were the cystic and common ducts. The head of the pancreas was enlarged and very hard. The gall-bladder was opened, when the wall was found to be infiltrated and the mucous membrane congested; no stones found. The duodenum was the seat of a thickened cicatrix of a healed ulcer in its first part just external to the pylorus. The gall-bladder was drained.

After an illness of about three months he succumbed to septic cholangitis.

CONCLUSIONS. 1. The coexistence of cholecystitis and duodenal ulcer is not rare, and in operating for gall-bladder disease a careful examination of the duodenum should always be made.

2. In this small series, although both conditions were present, the symptoms of gall-bladder disease predominated except in one case.

3. In making a diagnosis the clinical examination is much more reliable than the roentgen-ray.

4. This series would seem to confirm the view that multiple operations in the abdomen, as too frequently practised, are unwise and often unjustifiable.

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SETTLED AND UNSETTLED PROBLEMS IN GALL-BLADDER SURGERY

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The mortality connected with cholecystostomy and cholecystectomy will depend largely upon what operation is done in the serious complications of biliary disease, so in order to more clearly grasp the whole problem I think the causes of death in this group of operations should be thoroughly studied.

There are four outstanding contributory causes of death in this field: (1) Long-standing jaundice; (2) cardiac vascular renal disease; (3) abscess around cystic duct; (4) carcinoma.

The other chief contributory causes are: (5) Lung complications, including embolism; (6) associated pancreatitis; (7) acute hepatitis; (8) liver stones; (9) peritonitis.

My own mortality has been largely confined to this group. Most of these causes of death are so well understood it is unnecessary to more than mention them. Some of them, however, I think need further emphasis.

In studying the deaths among my cases I have been struck with the number apparently due to heart complications, particularly in aged people. Immediately it will be said that this is an easy way of avoiding the real cause of death, but the cases to which I refer for the most part came to autopsy. The picture soon after operation is one of acute cardiac dilatation; pulmonary edema, enlarged heart, with no abdominal symptoms. Some had more or less urinary suppression associated with edema, but I have felt the real condition was cardiac, because autopsy showed very little outside of chronic interstitial nephritis with cardiac dilatation. I am more and more impressed with the conclusion that certain hearts, particularly in the aged, which do not show any clinical evidence of cardiac disease sometimes are much more

impaired than some which do. Seldom is difficulty experienced with compensating hearts with clinical evidence of disease.

Much has been written regarding embolic processes in the lung, but I am convinced it is a much more frequent incident than we have heretofore believed. Many times one sees, from the second to the fifth day, cases developing pulmonary symptoms, usually with mild temperature, 100° to 101° F.; some cough, sometimes bloody; they usually recover. These are usually ascribed to the anesthetic, but I am sure they are embolic in character. Certainly pulmonary embolism is a much more frequent cause of pulmonary complications than usually supposed and is a definite cause of death in a certain percentage of cases. Massive collapse of the lungs I have not seen in any case or, at least, did not recognize it.

Abscess around the cystic duct is a very serious disease. I was amazed to read in Deaver's book it carries an operative mortality of over 30 per cent. These, of course, ultimately must have cholecystectomy done; but I wonder, in view of the very high mortality, if it would not be wiser to do a two-stage operation, contenting ourselves with mere drainage at the first operation?

As one studies this group, where the large percentage of deaths occur, one is surprised to find the number where the mortality is beyond our control. Any series of cases which include a fair proportion of cases in this group is sure to have a large death-rate, consequently any discussion regarding mortality in cholecystectomy and cholecystostomy is at least unscientific unless we know which operation is done in these complications of gall-bladder disease. In my own series of 500 cases cholecystostomy has a higher mortality, merely because I have used this operation in the complicated cases.

I am thoroughly convinced that in uncomplicated disease of the gall-bladder the mortality in both operations is so low it ceases to be a matter for controversy. The question has to be settled on other lines, such as permanent cures and the effect of both operations on diseases of the liver and pancreas.

Possibly we can start on a common ground by stating a few of the supposed "settled problems." I will mention a few of these, although with some of them you may disagree:

1. While infection may not be present inside the gall-bladder, it may exist in the wall of the gall-bladder. Experiments of

Rosenow, confirmed by many others, together with clinical experience seem to substantiate this opinion.

2. Stones recur after cholecystostomy.

3. The mortality is so low in uncomplicated diseases of the gall-bladder, with or without stones, in both cholecystectomy and cholecystostomy, that a decision in favor of one or the other should rest upon other factors.

4. Cholecystectomy is obviously the operation of choice in:

(*a*) Stones in the cystic duct: The frequency of stricture here, necessitating future cholecystectomy, would seem in the absence of abscess to make primary cholecystectomy imperative.

(*b*) Stone in the cystic duct with local abscess: Cholecystectomy is imperative, whether it be done in one or two stages of operation.

(*c*) All forms of gangrenous cholecystitis.

The "unsettled problems" might be subdivided into:

1. Those in which the majority of surgeons agree on the correct procedure.

2. Those in which the majority of surgeons are more or less in doubt.

In the first group I believe the majority of surgeons prefer cholecystectomy in:

(*a*) Adhesions around the gall-bladder, without other evidence of disease: I am sure I am not always able to tell what cases are due to disease from infection of the gall-bladder or to the so-called congenital adhesions. In the presence of clinical evidence of gall-bladder disease, I believe these cases should have a cholecystectomy done; on the other hand, I am sure some cases have congenital adhesions around the gall-bladder, which produce no symptoms. Here the gall-bladder should be left alone. I hope the discussion will shed further light upon how to distinguish between the normal and the abnormal gall-bladder in the presence of moderate adhesions without other evidence of disease.

(*b*) Gall-bladder with stones confined to the gall-bladder: There has been so much discussion upon this subject that I hesitate to bring it up at this time. From personal observation and from the literature, I am quite convinced the very large majority of surgeons today prefer cholecystectomy. I presume the smaller minority will continue to do simple drainage. The object of this paper, however, is to emphasize the importance of the complicated

case, rather than the uncomplicated, as I personally believe, in the absence of such complications as jaundice, old age, bad risks, etc., the question is almost settled in favor of cholecystectomy.

(*c*) Gall stones, with a low-grade biliary hepatitis: Much has been written of late on this subject regarding (1) its frequency and (2) the necessity of liver drainage in these cases. I have no doubt more or less of a low-grade liver infection is present in most cases of infected ball-bladders, but I am sure they are cured by cholecystectomy.

(*d*) With stones in the common duct and a contracted, non-functionating gall-bladder, cholecystectomy with common-duct drainage should be done, because the gall-bladder is useless for any subsequent procedures. Usually the gall-bladder is a small, contracted organ with a large amount of scar tissue, which if left can only be a source of trouble and of no possible benefit for future operative anastomosis.

In the second group, where I think surgeons are more or less in doubt, the decision seems to me to be much more difficult:

(*a*) In the common-duct stones with an apparently functioning gall-bladder, the 15 per cent Courvoisier's law, if we are sure all stones are removed from the common duct, cholecystectomy with drainage of common duct is a safe procedure. However, complications in these cases are so frequent that probably in most instances cholecystostomy with drainage of common duct is the operation of choice.

(*b*) Stones confined to the gall-bladder, complicated by jaundice: The jaundice I believe is due to an associated infectious cholangitis. I presume most surgeons would believe in simple drainage; but if we believe that tying off the cystic duct causes better drainage, by forcible dilatating the valve of Oddi, then cholecystectomy should be done. Personally, I am in doubt, but usually do a cholecystostomy when jaundice is present.

(*c*) Chronic pancreatitis with or without stones in gall-bladder: Again this question depends on the ultimate solution of the effect of tying off the cystic duct on the valve of Oddi. Chronic pancreatitis is a very unsettled problem. I think that Warthin's opinion, that these are largely syphilitic in origin, is an error, because we have treated many without improvement. I presume the severe grades of disease which simulate malignancy should be treated by drainage, either external or into the stomach or duo-

denum, while possibly the milder forms should have a cholecystectomy.

(*d*) Subacute pancreatitis with areas of fat necrosis is a very difficult problem. Of course, all gall-bladder complications should be treated in the usual surgical manner, but just what to do with the pancreas is a disturbing problem. I am inclined to believe that these mild, subacute pancreatitis cases might be divided, like the old adage, into those who get well and those who do not. Is the pancreatitis really due to associated infection in the biliary system or obstruction to the pancreatic ducts? If due to the former, I can see where biliary drainage in some form would be indicated; but if to the latter, I cannot see the purpose of it. Drainage of the lesser sac in the absence of localized areas of hemorrhage seems almost futile.

Much experimental work has been done to prove that these cases are due to obstruction. We carried on many experiments on cats and found that, although the cystic duct was quite large, the common duct was so fine it was almost impossible to force anything inside the duct. Consequently, I view with considerable doubt conclusions based on these premises. Is it not much more likely the disease is either metastatic from blood infection or, as Deaver views it, direct extension through the lymphatics?

The acute hemorrhagic forms of the disease are entirely different. Here the mortality is exceedingly high and the less done, with ample drainage, would seem to be preferable.

(*e*) Carcinoma of the pancreas and ampulla: If the diagnosis is certain, I feel those cases should not be operated upon. The difficulty of distinguishing between common-duct stones and carcinoma of the pancreas, especially early, is so great that operation is advisable in most instances for diagnostic purposes. If at operation the diagnosis of malignancy is positive, one has the choice of doing simple closure of the abdomen or some form of anastomosis between the gall-bladder and, preferably, the stomach. Cholecystostomy is, I think, a mistake. Patients almost invariably die much more rapidly than if no operation were performed.

In considering the question of hepatitis there seems to be a good deal of loose thinking. Of course, it necessarily follows if there are gall stones with an infected gall-bladder, there is probably some associated liver infection. In fact, the frequency with which actual stones form in the liver is not sufficiently emphasized.

I presume the term biliary cirrhosis is as good as any other, but I wish to emphasize that this is an entirely different pathologic entity from acute hepatitis. A biliary cirrhosis is cured by treating the cause, *i. e.*, disease of the biliary system, but acute hepatitis is not influenced by similar treatment, because the infection is proximal to the gall-bladder and ducts.

Some cases I have seen have been to me very unusual; in fact, I had never seen any until the past few years. They begin with fever, later jaundice and usually chills. The temperature varies from 101° to 104° F., and is the usual septic temperature. The chills have no regularity. Jaundice is almost always present to a varying degree. At operation the varying complications are present in the gall-bladder, but their treatment has no influence on the condition in the liver. The disease runs its course regardless of cholecystectomy or drainage, usually ending in death.

The liver has varied from a moderately large liver to a very small one. The pathologic reports varied. Dr. Roman in three fatal cases reports: (1) Interstitial hepatitis with jaundice of the liver parenchyma; (2) cholangitis with biliary cirrhosis; (3) acute cholangitis of the smaller intrahepatic ducts, degeneration of liver parenchyma and bile stasis with slight fibrosis and slight reduction of size of organ. Two cases were reopened after several weeks on the advice of the medical attendant, thinking stones were overlooked in the common duct. They had chills; temperature at irregular intervals simulating common-duct stones. Both cases showed very extensive adhesions all around the liver, but no stones were found.

The whole pathology of liver infection seems at best poorly understood. It would seem as if at one end we had a mild biliary cirrhosis and the other an acute yellow atrophy with varying grades, including infectious jaundice, intervening.

Finally, should we not view the whole subject of biliary surgery with a broader vision than simply a conflict between cholecystectomy and cholecystostomy? The mortality in both is so small in the uncomplicated case that it need not be considered. The real problem is, if possible, to definitely establish the correct procedure in the associated complication of duct, liver and pancreatic disease.

THE SURGICAL MANAGEMENT OF THE COMPLICATIONS OF CHOLECYSTITIS

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Cholecystitis usually begins as a chronic insidious process, the inaugural symptoms of which are disregarded by the patient and may escape the attention of the physician. Recognition of the early symptoms and prompt surgical treatment secure the best results with a minimum of risk. The recurrence of symptoms in a definitely small percentage of cases after cholecystectomy is usually the result of preëxisting complications. The surgical mortality of cholecystitis and its complications walk hand in hand. Cholecystectomy is a highly satisfactory operation because when performed early it removes the disease.

Cholecystostomy is and always will be a useful operation, not because it saves the gall-bladder, but because it is a simple and safe method for relieving acute symptoms and providing drainage.

Acute, subacute and chronic cholecystitis are, as a rule, readily differentiated; but discrepancy between the clinical and pathologic findings is not uncommon and the diagnosis of acute cholecystitis is often made only after the abdomen has been opened. A patient may be acutely ill with fever and prostration, following an attack of pain in the upper right abdomen, and at operation the pathology disclosed does not adequately explain the symptoms. Sometimes with mild symptoms and no constitutional disturbance the exploration discloses intense inflammation of the gall-bladder with edema and many fresh adhesions. In a third group the systemic reaction with local pain, tenderness, rigidity and sometimes a palpable mass, point unmistakenly to acute

cholecystitis, and the diagnosis is confirmed by the pathology disclosed. Acute cholecystitis is surgical, but when possible operation should be deferred until the subsidence of the acute attack. During the acute stage there is likely to be a general portal infection, which simmers down, to localize in the gall-bladder. The surgeon, however, must be on the alert to detect any untoward symptoms indicative of perforation or suppuration which will demand immediate interference. Operations carried out on the acutely inflamed gall-bladder—which is white, distended, thick, edematous and surrounded by adhesions—require accurate and thorough technic. Whenever possible, such a gall-bladder should be removed, for, as in a case of acute inflammation of the appendix, it gets rid of the disease. The operation is tedious, because of inflammatory exudate around the neck of the gall-bladder, which obscures the landmarks and predisposes to traumatic injury of the main ducts. By cutting through the tissue just below the neck of the gall-bladder, the cystic artery and cystic duct are divided and can be picked up separately and ligated. They do not retract because of the surrounding inflammatory exudate. Of course, when these structures can be accurately identified it is better to clamp them before cutting. By careful dissection, dividing the anterior layer of the gastrohepatic omentum overlying the common duct, the latter is followed up until the cystic duct is exposed, when it is clamped with two long, narrow forceps and divided between the clamps with the cautery or scissors. The cystic artery is next exposed, clamped and cut. In the event of a long cystic and long hepatic duct, the two joining just short of the duodenum, or the presence of an anomalous cystic artery, this dissection will expose them, and will also avoid injury to either the common or the hepatic duct. It will now be found that the gall-bladder can readily be peeled from its attachment to the liver.

Gangrenous cholecystitis is very rare, because the gall-bladder has a rich blood supply through the cystic artery and the numerous vessels in its attachment to the liver. In acute cholecystitis the main bloodvessels in the wall of the gall-bladder may become thrombosed and gangrene may ensue. This condition, when it occurs, is usually an outcome of acute cholecystitis and requires similar treatment.

Acute perforation of the gall-bladder is unusual, because of the

protection afforded by surrounding structures, and because bile tension within the gall-bladder is never high. The symptoms resemble those of perforated peptic ulcer, and immediate operation is equally imperative. The wall of the normal gall-bladder is strong and capable of marked distention. When diseased, the muscle fibers and connective tissue are invaded and weakened by inflammatory products. There is proliferation of the intramural glands and deepening of the crypts which honeycomb the wall. These changes predispose to slow perforation and permit the formation of surrounding adhesions, with frequent attachment to the duodenum, stomach or colon. Finally, a communicating fistula may form. Attachment of the gall-bladder to the duodenum is more often caused by cholecystitis than by duodenal ulcer. When the gall-bladder is found adherent to a neighboring viscus a careful search should always be made for a fistulous opening, which may be very small. In case the fistula is large and patulous, there is a strong probability of an obstructing stone in the common duct. The best procedure is, when possible, to remove the gall-bladder and close the fistula, and make a careful exploration of the common duct for stone.

Calculi are usually the result and sequel of cholecystitis. Perversion of cholesterin and fat metabolism may predispose to their formation. Modern opinion favors infection of the gall-bladder as the primary lesion. The cardinal symptoms of gall stones and cholecystitis are often identical. Calculi may form early, and are nearly always present in the late or advanced stages of cholecystitis. According to our conception of the pathology of cholecystitis, it is not reasonable to expect that inflammation and infection will disappear when only the stones are removed. The patients who have had cholecystostomy for stones may, and often do, continue to have persistent cholecystitis, although they may be temporarily relieved of symptoms, but more stones often reform. The cystic duct and the neck of the gall-bladder are usually involved in cholecystitis and are frequently obstructed. Permanent relief of symptoms by simple removal of the stones presupposes that the gall-bladder is capable of returning to its normal condition and that the cystic duct is patulous. But the former does not occur, and the latter is not always the case. In most instances Nature has already extirpated the gall-bladder. The indications for cholecystostomy will be determined by the

individual case, and will depend on the necessities of risk, local pathology and additional complications, such as acute hepatitis, cholangitis with stone in the common duct and pancreatitis. In short, we believe that in cholecystitis, with or without stones, the gall-bladder should always be removed when conditions permit, unless it is to be used for biliary drainage, and even here drainage of the common duct is often more satisfactory. Retention of the gall-bladder for utilization in secondary operations on the biliary tract will be less indicated as the primary operation is made more complete and thorough.

The essential significance of jaundice is a disturbance of that part of the hepatic function which has to do with the elimination of bile pigment. In this respect the liver resembles the kidney in its relation to urea. Therefore, jaundice may be functional or obstructive. The functional type may depend on the inability of the diseased liver to excrete its normal allotment of pigment, or, in case the formation of pigment is excessively rapid, the normal liver cannot adequately dispose of it. The latter condition probably prevails in hemolytic jaundice. The liver probably modifies in some way the pigment brought to it. In the obstructive type of jaundice the block may be anywhere in the duct system, either within or outside the liver. Intrahepatic obstruction may be the result of inflammation and edema of the smaller ducts. Extrahepatic obstruction is caused by stone, stricture, carcinoma and pancreatitis, and clinically is frequently complicated by infection. Obstruction of the common duct not only prevents the exit of bile, but also impairs the activity of the liver, so that in this condition functional and obstructive jaundice may be coexistent. Cholecystitis may be a cause of functional jaundice, but repeated attacks of colic and jaundice are usually caused by stone in the common duct.

Within the past few years the mechanism and pathologic significance of jaundice have been in large part explained, and a method has been devised for measuring the pigment content of the blood. Bile pigment is formed in large part outside the liver, and carried to it for excretion. Failure of excretion causes pigment to accumulate in the blood, and when the quantity rises above the threshold value of the kidney it appears in the urine and later passes out of the bloodvessels into the tissues. Therefore, in biliary obstruction the degree of jaundice is dependent on

variable factors, namely, the extent of the obstruction, the threshhold value of the kidney, its capacity to excrete pigment and the permeability of the bloodvessels to pigment, so that jaundice becomes an imperfect manifestation of increased pigment content of the blood.

The quantitative test for bile pigment in serum is particularly useful for following the progress of jaundice and as a guide to the safest time for operation, but its interpretation must consider the above factors. The capacity of the kidney to eliminate pigment may explain the varying tolerance of patients for jaundice. The liver is the main site for urea formation and bile excretion; while the kidneys are the main exit for the former product and in obstructive jaundice, they attempt to take over the latter function. Long-continued jaundice produces a toxic nephritis, with an accumulation of urea as well as pigment in the serum. In jaundice there is a well-known tendency to bleed. In our experience this tendency cannot always be measured by the coagulation- or clotting-time of the blood, and is not always directly proportional to the intensity of jaundice. We have noted that any tendency to bleed rapidly disappears when bile drainage is established.

Surgical mortality in jaundice is probably more often the result of failure to establish bileflow, and of hepatic and renal insufficiency, than from actual loss of blood by oozing. The danger factors in obstructive jaundice are pigment and bile salts in the blood, tendency to bleed, liver insufficiency and kidney insufficiency. To combat and control these factors should be our aim in the preoperative preparation of the patient. It is our practice, after careful study of the patient and employment of the recognized laboratory tests, to build up the glycogen reserve of the body by the administration of glucose and insulin, to supply adequate fluid intake and to fortify the blood by injections of calcium chloride and by blood transfusion. One must be cautious not unduly to prolong the period of preparation, and thus miss the opportune time for surgical intervention. Here the judgment and experience of the surgeon are the best guides. Cases of painless jaundice are the ones most suitable for study and preparation. When the symptoms, such as pain, jaundice and intermittent fever, indicate a stone in the common duct, there is little to be gained by the usual methods of preparation; prompt

surgical treatment is the best procedure. Operations on jaundiced patients should be deliberate and thorough. The practised surgeon will readily detect the basic pathologic lesion. The operation should be completed when possible in one sitting rather than divided into steps. Secondary operations on the biliary tract are not desirable, either from technical or from therapeutic standpoints. To drain the gall-bladder or common duct, and intentionally leave a stone for later removal, is not for the best interests of the patient. In most instances the diseased gall-bladder and the stone in the common duct can be removed safely at the same time. After this procedure we drain the common duct with a T tube. When obstruction is caused by disease of the pancreas, and the gall-bladder is dilated and tense, internal drainage and cholecystoduodenostomy is the method of choice.

There is abundant clinical data to support the belief that pancreatitis is most often a sequel of cholecystitis; we believe that the usual pathway of infection is through the lymphatics. It is difficult to prove or disprove this by experiment; we must rely on pathologic and clinical deductions and on the testing ground of experience at the operating table. We do not deny that the pancreas may at times be the primary seat of infection or that disease may reach it by other channels than the lymphatics from the gall-bladder. A felon on the finger may be simply a local affair; it may involve the hand and arm through tendon and fascial planes; it may reach to regional lymph nodes and cause painful swelling or suppuration; or within a few minutes it may blaze up the arm as a virulent lymphangitis. A similar relation may prevail between cholecystitis and pancreatitis, from the mild and chronic to the suppurative and ultra-acute forms. Acute pancreatitis has clinical and pathologic varieties, which determine the type of treatment and the mortality. Some of the mildly acute forms, even in the presence of fat necrosis and free fluid, will subside with or without surgical intervention. Some of the suppurative types yield to simple incision and drainage when localization has been effected. Most of the ultra-acute cases speedily perish regardless of the course of treatment pursued. The clinical symptoms cannot always be relied upon to differentiate the types of acute lesions. In the management of acute pancreatitis, when the onset has been with agonizing epigastric pain, accompanied by the signs of an abdominal catastrophe, it is

our practice to operate at once. The pancreas is exposed through the gastrocolic omentum and, if it presents no gross lesion, drainage is carried down to it. If there is an abscess or a hematoma the gland is incised with a blunt instrument, and drainage provided for by gauze tampons surrounded by thick rubber dam and a rubber tube. In the subacute varieties immediate operation is not so imperative, because a localized abscess often forms, which can later be drained through the abdomen and sometimes by an extraperitoneal approach through the left loin.

The pancreas may be affected by chronic inflammation, independent of disease of the biliary tract, but in the surgeon's experience chronic pancreatitis is most often a sequel of cholecystitis. The head of the gland, the part embraced by the curve of the duodenum, is the usual area of involvement. In spite of the intimate relation between this portion of the gland and the common duct, biliary obstruction rarely occurs when the pancreas is enlarged and soft. Drainage of the gall-bladder, except when the condition is very acute, will not have much effect upon the pancreas. Cholecystectomy, by removing the focus of infection, accomplishes the most good. When biliary obstruction is present the head of the pancreas will usually be found enlarged and hard, and often is indistinguishable from carcinoma. In these cases the pancreatic condition is probably not secondary to cholecystitis, because in the absence of carcinoma cholecystoduodenostomy is usually followed by permanent relief of symptoms. Cholecystostomy may be made, but biliary drainage must be protracted and has obvious disadvantages. We have seen a few cases of pancreatitis following cholecystectomy when a small stone has been overlooked in the terminal portion of the common duct.

Stone in the common duct is a complication of cholecystitis, because, with few exceptions, all stones originate in the gall-bladder. The condition should be extremely rare after cholecystectomy, unless a stone has been overlooked at the original operation, which unfortunately may occur. In the removal of the gall-bladder which contains stones it is important to be sure that small stones or sand are not allowed to remain behind in the stump of the cystic duct, because of the danger of their ultimate entrance into the common duct, where they may lodge. A similar sequence of events may occur in cholecystostomy when small

stones in the cystic duct are very likely to be overlooked. The prevention of stones in the common duct, in addition to the above precautions, obviously consists in early removal of the diseased gall-bladder.

From the clinical standpoint, choledocholithiasis may be frank or obscure. The deception of the silent stone, so-called, varies with the audition of the surgeon. The classic syndrome of colic, jaundice and rigors is well known. Often one, sometimes two, of this cardinal trinity may be absent. Careful palpation of the common duct should be a routine procedure in all operations for cholecystitis. The features which cast suspicion upon the common duct are a history of jaundice following repeated attacks of colic, a contracted gall-bladder and a dilated, thick-walled duct. It is not necessary to open and explore the duct in every suspicious case unless careful inspection and palpation throughout are prevented by the presence of exudate or adhesions. However, no doubt must remain in the mind of the surgeon. The principle of thoroughness is more important in this special field of surgery than in almost any other.

Stones in the supraduodenal portion of the common duct are easily removed. The duct may be grasped by forceps on each side and incised for about ½ inch; following a gush of bile the stone often pops into the wound. The duct should then be explored with the finger, if possible, or with instruments which are made to pass into the duodenum. When the stone removed is facetted, look for more stones. Sand and puttylike material are best removed with a scoop. Flushing the duct is apt to carry stony débris into the intrahepatic ducts. When a stone is impacted in the ampulla, or in the retroduodenal portion of the duct, it may be milked back, or, if necessary, crushed and removed piecemeal. We rarely find it necessary or advisable to open the duodenum, although we sometimes mobilize it to expose the stone. Thoroughness is essential and the lumen of the duct must be cleared into the duodenum. We always drain the duct with a T tube and, when possible or advisable, remove the gall-bladder. The lower arm of the T tube is purposely not allowed to enter the duodenum, because to do so accomplishes no good and may favor ascending infection. The T tube is allowed to remain for at least three weeks, sometimes longer.

The advantages of preoperative preparation of patients with

jaundice caused by stone in the common duct are usually exceeded by the risks of delay after the diagnosis has been established. In the absence of cholangitis, and when subsidence of jaundice indicates relief of biliary obstruction, it is advisable to await the optimum improvement of conditions.

It is sometimes difficult to differentiate between biliary obstruction caused by stone and by carcinoma or stricture. However, calculus obstruction is usually painful and incomplete, and the jaundice is variable and often accompanied by fever. In about 20 per cent of cases of stone in the common duct jaundice is absent.

Hepatitis denotes inflammation of the parenchyma of the liver and has been shown to be most constantly associated with cholecystitis. In the acute form there is diffuse, dull, aching pain over the liver, and sometimes fever and jaundice. The liver appears dull red and mottled; it is enlarged and the edge is rounded. In the chronic type the part of the liver adjacent to the gall-bladder is grayish-white, contracted and firm, the result of fibrous tissue. The present interest in hepatitis is chiefly from the standpoint of pathology and its relation to the etiology of cholecystitis. Little is known of its ultimate effects. Marked chronic hepatitis may exist with only slight changes in the gall-bladder, and in advanced destructive cholecystitis the liver may be grossly normal. Present surgical opinion holds that when there is visual evidence of hepatitis the gall-bladder should be removed.

Varying degrees of cholangitis are probably always associated with cholecystitis and with stone in the common duct. In acute or suppurative cholangitis the surgical procedure should be the least possible consistent with immediate relief, which is best secured by biliary drainage.

Regarding the subject of biliary cirrhosis, we are in accord with the principles so ably expressed and advanced by W. J. Mayo. The condition is usually a late complication or sequel of chronic cholecystitis and cholangitis, and often follows the intermittent biliary obstruction from a stone in the common duct. Cholecystectomy and removal of the obstruction may provide relief, but in well-established cases there is often persistent mild jaundice, which sometimes clears up after splenectomy.

Cholecystitis affects the period of life when the attainment of physical maturity passes into the phase of depreciation; when the

active processes of growth are being replaced by the struggling efforts of repair and maintenance. In the battle with infection several structures may fall together, or the fall of the one may weaken the defence of the other. The association of lesions, a league of lesions if you will—we speak of cholecystitis, peptic ulcer and appendicitis—is actually so frequent as to be common surgical knowledge. Clinical experience leads us to believe that these gastroenteric lesions follow in sequence from a primary focus of infection, which is usually in the appendix. It would be of interest to compare the relative incidence of cholecystitis or ulcer in patients who have had their appendix removed with those who still retain it. It is our impression that many patients who are operated on for cholecystitis or ulcer subsequent to appendectomy had the above lesion prior to removal of the appendix. We advocate and practise in all abdominal operations for chronic disease examination of the gall-bladder, stomach and duodenum, by sight and touch, whenever possible, and we usually remove the appendix.

The causes of recurrence of symptoms following operation on the gall-bladder may be grouped as follows:

1. Incomplete primary operation, such as failure to remove the diseased gall-bladder, the appendix and a common duct stone, or to recognize and treat a peptic ulcer and chronic pancreatitis. Cholecystostomy may temporarily relieve the symptoms of, but does not cure cholecystitis, and by its mechanical effects may be the cause of new symptoms. Likewise, persistence of symptoms after gastroenterostomy for ulcer may be caused by cholecystitis. Removal of the appendix, even when not acutely diseased, is a safeguard against future trouble.

2. Hepatitis and pancreatitis may persist for several months after cholecystectomy, and then subside. On several occasions we have drained the common duct in these cases with good results.

3. Accidents of the operation, of which injury to the hepatic or common duct is the most serious, and unless promptly recognized and treated may result in a stricture with jaundice or a complete biliary fistula.

4. Incorrect diagnosis, both before and during operation. The surgeon must be trained in the art of diagnosis and in the recognition of pathologic tissue.

AN ANALYSIS OF GALL-BLADDER CASES, FROM THE FIRST SURGICAL DIVISION OF THE NEW YORK HOSPITAL

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AND

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Any analysis of a group of cases must necessarily present a mass of figures more or less statistical. It is not the intention of this paper to try to prove anything by these statistics. The cases represent the ordinary run of patients who present themselves for treatment in a metropolitan hospital of the type of the New York Hospital, and represent a varying racial type as well as a great variety of pathological conditions in the gall-bladder.

Our follow-up system, instituted by Gibson, began in 1913, but was not in complete working order until 1914, and the cases used for the basis of this paper are those from 1914 to 1924, inclusive.

The principal procedure in treating gall-bladder disease on the First Surgical Division has been cholecystectomy, when that procedure has been deemed advisable. In a very small group of cases this has been the so-called "ideal cholecystectomy," with peritonealization of the gall-bladder area and the closure of the abdomen without drainage. In case of any doubt in the operator's mind, drainage has been instituted.

In the cases not suitable for cholecystectomy, that is, in cases which were too seriously ill, or whose general or local condition did not warrant the more radical procedure of cholecystectomy, and in those cases with marked pancreatic changes which required prolonged drainage, cholecystostomy was the method of choice. This was later followed by cholecystectomy if the result thus obtained was not satisfactory.

Use of Various Tests. The icteric index has shown biliary retention, but so far as our series goes, has not been of any prognostic value either as to the outcome of the operation or as to the

result obtained from the operation. The Graham test has proven of diagnostic value. Its use has been confined to too small a number of cases in this series to include the figures for the result of that test.

The choice of the operation must be left to the operator and made to fit the case, and, in so far as it is possible to say, such was the procedure in the list of cases here reported. The major portion of the cases were operated upon by the senior members of the staff, with a scattering number of cases among the junior members, and the result represents, we believe, a very fair presentation of the outcome of surgical procedure for biliary disease in the hands of a competent surgical staff, with its material derived from the ordinary ward cases in a city hospital.

In the follow-up certain terms are used to express the result. A case is classified as *excellent* when the patient is relieved of all symptoms, has no complaints and in which the result of the post-operative examination shows a practically perfect result from the operation.

The term *satisfactory* is applied to those cases in which there is some minor complaint or some finding in the examination which does not place the patient in the excellent class, but in which the result is, from the surgical standpoint, considered a satisfactory one.

The cases are classed as *unsatisfactory* chiefly from the story given by the patient of his condition following the operation. Many of the cases so classified have little, on physical and *x*-ray examination, to justify many of the complaints which these patients have, but it has been our feeling that some distinction had to be made and in the long run we could strike a better average by keeping to the above three classes rather than by making a more elaborate classification. After all, the patient must be considered and his complaints are an index of the benefit derived from the treatment.

Our death-rate may seem high, but if the deaths are analyzed it will be evident that the outcome could not be improved, considering the character of the cases in question and the condition of the patient at the time he entered the hospital.

During the period given above 482 cases of gall-bladder disease have been submitted to surgical treatment. Of these 482 cases, 400 were submitted to cholecystectomy and 82 to cholecystostomy.

RESULTS.

	Cholecystectomy.		Cholecystostomy.	
	No.	Per cent.	No.	Per cent.
Excellent	205	50.75	32	39.02
Satisfactory	112	28.00	20	24.39
Unsatisfactory	30	7.50	13	15.85
Dead	39	9.75	13	15.85
	(29	7.20)	(8	9.70)
Not found	16	4.00	4	4.87
	400	100.00	82	99.98

From the above, 78.75 per cent of the cases of cholecystectomy have given a satisfactory surgical result, that is, the patient has been cured; 63.41 per cent of the cholecystostomy cases also come under the same category. There is a higher group of unsatisfactory cases in the cholecystostomies, and, as would be expected, a higher mortality, because that procedure was used in the poorer surgical risks.

In analyzing the deaths in the list of cholecystectomies, 2 of the cases died at home before the three-month period, after leaving the hospital, of conditions not known but which the family stated had come from the operation. Of the 37 cases which died in the hospital:

	Died.
Carcinoma of the gall-bladder	3
Shock	5
Peritonitis	7
Cause not known	1
Pneumonia and bronchopneumonia	6
Pulmonary embolism	2
Carcinoma of the stomach	1
Acute nephritis	1
Acute cardiac failure	2
Acute pancreatitis	1
Stenosis of common duct	1
Septicemia	1
High temperature—cirrhosis of the liver	1
Acute psychosis	1
Hemorrhage	1
Constant hiccough	1
Myocarditis	1
After secondary operation in which echinococcus cyst of the liver was found	1
	37

In the deaths, peritonitis (7), shock (5) and pneumonia (6) occupy the leading positions. Three of the cases which died of shock had choledochotomy for common-duct stone. If the cases such as those with carcinoma of the stomach, carcinoma of the gall-bladder, acute pancreatitis, cirrhosis of the liver, echinococcus cyst and the cases with choledochotomy are eliminated, the mortality for cholecystectomy will be reduced by 10 cases, leaving a total death rate from cholecystectomy of 7.2 per cent.

Of the cases submitted to cholecystostomy, 13, or 15.85 per cent, died:

	Died.
Carcinoma of gall-bladder	3
Secondary operation for echinococcus cyst of the liver	1
Acute pancreatitis	1
Shock	2
Pulmonary embolism	1
Cardiac failure	1
Asthenia	1
Chronic pancreatitis	1
Lobar pneumonia	1
General peritonitis	1

Three cases classed as unsatisfactory died after leaving the hospital: 1 carcinoma of the liver, 1 carcinoma of the lung and 1 carcinoma of the stomach.

Here again many of the conditions for which the operation was undertaken might well be removed from the list of fatalities of cholecystostomy. Thirty-five cases had choledochotomy for common-duct stone with 3 deaths, 9 per cent, with 28 excellent results. Four cases were unsatisfactory and required secondary operations, 3 for stenosis of the duct and 1 for persistent biliary fistula.

In the cases in which gall stones were present in the gall-bladder the result of the follow-up showed a higher percentage of excellent results than was true where cholecystitis alone was present:

	Excellent, Per cent.	Unsatisfactory, Per cent.
Gall stones	48	5.7
Cholecystitis	41	9.9

The cases with carcinoma of the gall-bladder and duct all died.

The age incidence of the onset of gall-bladder symptoms is also interesting in that more than half the cases had symptoms of gall-bladder disease before the fortieth year, and in many cases

over forty. The history gave digestive disturbances for many years before the onset of the symptoms, sufficiently definite to cause the patients to seek medical advice, that is, our cases show an earlier age incidence than that given for gall-bladder disease. The youngest case had symptoms at the eighth year, and was operated upon when a little over ten years of age and a large gall stone removed with a pathlogical gall-bladder.

The intercurrent diseases or conditions found and treated were:

	Cases.
Fibromyomata of the uterus	33
Salpingitis, chronic	9
acute	1
Appendicitis, chronic	105
acute	1
Abscess, pelvic	1
tuboövarian	1
Oöphoritis	1
Endocervicitis	1
Endometritis	1
Pregnancy, tubal	4
Hernia, umbilical	6
inguinal	2
Ovarian cyst	7
Retroversion and retroflexion of uterus	6
Intraligamentous cyst	3
Nonrotated cecum	1
Pancreatitis, chronic	26
acute	1
Ventral hernia	2
Harris band	8
Diverticulum of the duodenum	1
Duodenal fistula	1
Cirrhosis of the liver	2
Interstitial hepatitis	2
Lipoma	2
Cervical polyp	1
Carcinoma of the stomach	1
Perforated ulcer of duodenum	3
Pyloric stenosis	2
Splenomegaly	2
Jackson's membrane	4
Duodenal ulcer	1
Gastric ulcer	2
Myosarcoma	1
Riedel's lobe of liver	1
Echinococcus cyst of liver	1
Angioma of ileum	1

STRICTURE OF THE COMMON DUCT

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Secondary operations on the biliary tract are among the most difficult tasks in surgery. The difficulty depends not only on the technical problems, but also on the presence of biliary cirrhosis and cholangitis. Stricture of the common duct always results in jaundice, either constant or intermittent. It is usually operated upon as a secondary operation, although the belief is becoming more prevalent, through accumulated experience, that in a considerable proportion of cases common-duct stricture is the result of a general obliterative cholangitis.

Considering these cases from the standpoint of management and treatment, they naturally fall into three groups: (1) Those in which the stricture is due to obliterative cholangitis; (2) those in which trauma at a former operation resulted in a complete biliary fistula; (3) those in which the stricture also results from operative trauma, but, on account of the closure of the ducts, is associated with complete and persistent jaundice.

Obliterative Cholangitis. In reviewing the records of our cases of stricture of the common duct, 64 in all, I have been greatly impressed by the fact that in a goodly proportion the common duct had been patent for many months, and in some instances for several years, after the cholecystectomy and before any sign of stricture developed. It is difficult to attribute these late manifestations to anything that took place at the time of the cholecystectomy. From some of the case records it is also found that there was clinical evidence of intermittent obstruction of the duct before any operation was performed. No cause could be found for this when the gall-bladder was removed, but in the absence of a stone it must mean that cholangitis was present prior to any operation. In certain other cases the signs of obstruc-

tion of the common duct have occurred soon after the gall-bladder operation; at the time of the secondary operation, instead of the expected stone in the common duct, an obliterative process was found extending throughout the course of the duct. Diffuse cholangitis is difficult to recognize because the inflammation in the walls of the ducts produces a great amount of edema in the duct and all the surrounding tissues, and the duct itself lies concealed in a hardened inflammatory mass. The disease for which the gall-bladder was removed is a part of the same obliterative process which extends through the liver and ducts.

Stricture of the common duct is frequently spoken of as congenital and acquired. It was formerly thought that the congenital type was the more frequent. Moynihan said that congenital stricture of the common duct is a part of the disease which has been described as congenital obliteration of the bile ducts. He thinks it is probable that, as Ralston has suggested, the disease is primarily instituted during fetal life by poisons derived from the mother and conveyed to the liver of the fetus, and that a combination of cirrhosis and cholangitis is set up. The result is an obliterative cicatrization of the ducts. This would seem to explain the truly congenital stricture found in infants shortly after birth and the absence of a demonstrable common duct at operation. It seems to me that this same process could be started in an adult by certain conditions independent of any inheritance. The literature has recorded many cases which have occurred between the ages of nineteen and sixty years. Usually these are given as single case reports, but all of them are similar in the clinical symptoms and findings. The stenosis comes on insidiously, and is accompanied by the usual signs of stone in the common duct. There is usually a complete remission between attacks. Frequently a number of small stones are found in the ducts, but these are probably the result of the stenosis and not the cause of the general obliterative process. A stone wedged tightly in the common duct might produce an ulcer, the healing of which would result in stenosis, but it is not likely that it could be the cause of a general obliterative process in the intrahepatic ducts as well. In my experience the occurrence of stricture following the passage or removal of stones from the common duct has been very infrequent.

Stricture has followed severance of the duct during the per-

formance of cholecystectomy, and undoubtedly some of those that are seen are due to this mishap. I am of the opinion that an obliterative inflammatory process throughout the ducts is the real cause of many of the strictures attributed to trauma. In 16 of the series of 64 cases, in which operation was performed for stricture, the condition was the result of obliterative cholangitis; in 15 more the etiologic factor may have been cholangitis. When the stricture is of this origin the obstruction is usually not complete, and, as the structures are separated from the surface of the liver and finally the common duct is opened, there is not the rush of bile or white fluid that is observed in the cases of traumatic stricture. The duct appears as a rounded opening on the surface of the liver and no amount of dissecting will isolate a common or hepatic duct. These are the most difficult cases of all to handle. The best that can be done is to make an opening in the duodenum and make a side-to-side anastomosis over a tube to the remnant of duct wall in the liver. The immediate results are usually fairly satisfactory, but in many of these cases the trouble recurs. It is not a good plan to advise another operation at the first sign of difficulty, as some of these patients will have slight attacks of chills and jaundice intermittently for a time and then become entirely well. In the event that the difficulty persists, further operation should be considered, as some of the most complicated conditions may finally be relieved, apparently as the result of subsidence of the obliterative inflammatory process. If the condition is not relieved, however, increasing biliary cirrhosis and destruction of hepatic tissue are sure to ensue.

Case I.—A woman entered the Mayo Clinic in November, 1912, at which time cholecystectomy was performed for chronic cholecystitis which had been causing symptoms for one year. She was relieved of all of her symptoms for two years following the operation, and then experienced vague and indefinite pain in the epigastrium which lasted for five days. After this attack she was free from symptoms for two years. At this time she began to have severe attacks of colic, usually accompanied by jaundice, and at the end of a year an exploratory operation was deemed advisable. A stricture was found at the juncture of the cystic and common ducts, incised, and the duct reconstructed over a T tube. Bile drained for seventeen days. The patient convalesced promptly. According to a report five years after the second operation, the patient is perfectly well and neither the pain nor the jaundice has recurred.

Comment. The stricture in this case was localized, but it did not occur until four years after the gall-bladder was removed. I believe that the best explanation is that the stricture was a part of an obliterative cholangitis.

Case II.—A woman, aged sixty-six years, first entered the clinic in December, 1911, at which time she gave a twenty-year history of epigastric pain, radiating posteriorly and to the right shoulder, and of indigestion and qualitative food distress. During the immediate past few months she had suffered from pain sufficiently severe to require morphin for relief, the attacks occasionally being accompanied by jaundice. At the first operation, in December, 1911, partial cholecystectomy (three-fourths) had been performed and a stone removed from the common duct. A Robson drain was used. The gall-bladder was empyematous and filled with stones. Bile drained for about two weeks, but after that the wound healed promptly and the patient remained well and free from symptoms for thirteen years. She returned to the clinic in 1922 because of myocarditis, rheumatic endocarditis and aortic insufficiency with acute decompensation. After medical treatment in the hospital for three weeks she recovered sufficiently to warrant her return home. She had no complaint that could in any way be attributed to the biliary system.

Four years later she again returned to the clinic; this time because of severe attacks of pain in the right upper quadrant of the abdomen, radiating posteriorly and requiring morphin for relief. Each attack was accompanied by nausea, vomiting and jaundice, but there had been no chills nor fever. She had had four attacks in two years. The heart showed some fibrillation and decompensation. However, the history pointed to abdominal pain of such severity that further operative treatment seemed advisable, and on February 11, 1926, an exploratory operation was performed. There was a small stump of gall-bladder remaining. The common duct was constricted at the juncture of the cystic and hepatic ducts. This stricture was incised, and the duct reconstructed over a Robson hepaticus drain. Some bile drained for about two weeks and then the wound closed. The immediate convalescence was satisfactory, and at the last report a month ago the patient stated that she was completely cured.

Comment. In this case the common duct had been patent and the patient free from all symptoms referable to the biliary system for thirteen years after the partial cholecystectomy and removal of the stone from the common duct. Whether a stricture occurring after that length of time was due to the ulceration where

the stone formerly lay, or whether it was due to an independent inflammatory process, is difficult to decide, but it would seem that it would be just as likely to be due to the latter as the former. In the few other cases in which a stricture has formed after a stone has passed or has been removed from the duct, it is possible that the stricture resulted from cholangitis.

CASE III.—A married woman, aged forty-one years, first came to the clinic in 1920, complaining of intermittent chills, fever and jaundice since cholecystectomy had been performed, in 1919. Her mother had died of cancer; no other family tendencies were elicited. The patient had been pregnant twice.

A few months following the cholecystectomy, in 1919, all of her symptoms had returned; the common duct had been explored, but no calculi were found. A tube inserted into the common duct drained bile for several months. After the tube was taken out, pain, chills and fever returned. The patient was jaundiced at the time of examination, in 1920. I performed hepaticoduodenostomy, using a large rubber tube. After this the patient did very nicely for four years, then had an attack of sharp pain in the right upper quadrant, with chills, fever and jaundice, which subsided only to recur at intervals, although soreness in the right upper quadrant had been constant since the first attack.

In January, 1926, five years after hepaticoduodenostomy was performed, the patient returned to the clinic. The hemoglobin was 49 per cent; erythrocytes, 3,190,000; coagulation-time, seven minutes. The serum bilirubin was estimated at 2.5 mg. for each 100 cc. of blood. The hepatic function test showed dye retention, 2. The roentgenogram showed the tube in good position in the duct.

February 3, 1926, I removed the tube from the intestinal tract. March 1, the patient was dismissed, apparently in good condition. The jaundice had entirely cleared up and the wound was well healed.

Comment. This case illustrates two points: (1) It seems to show that we are justified in continuing to operate if the trouble recurs; (2) that this woman was entirely free from symptoms for the four years that the tube was in place. One stitch had been used to hold the tube to the duct structures, but other than this nothing was done to keep it in place. After four years of relief she began to have symptoms of cholangitis and slight infection. It seemed best to remove the tube because it might be the source of this trouble. When it was removed it was thickly coated with

bile pigment. A small nick was made in the intestine, the tube withdrawn and the opening closed. There was some evidence of cholangitis for a time after this and it was three weeks before the jaundice had entirely disappeared.

Stricture of the Common Duct with Biliary Fistula. The cases of stricture in which there has been a biliary sinus and no jaundice are the more favorable for operation. Biliary cirrhosis and jaundice are not present. The chief difficulty lies in the handling of the duct which has not become dilated. It is well to keep in mind that in these cases, in spite of the biliary drainage and the lack of clinical signs of jaundice, there may nevertheless be a certain amount of biliary cirrhosis and latent jaundice, and careful estimates of the serum bilirubin should be made to determine the necessity for preparatory treatment before an operation is undertaken. In cases of biliary fistula the fistula, after dissection, can be used as a duct, and anastomosed to the duodenum, as has been done by Murphy, Lahey and others. Stricture does sometimes ensue, and it is often better to excise the fistula and anastomose the common or hepatic duct to the duodenum. Anastomosis of the stump of the common duct to the side of the duodenum has given the best results in my experience in all cases of stricture of the common duct. Sometimes it seems best to perform a plastic operation at the site of the stricture or an excision and an end-to-end anastomosis. If there is any trouble in locating the distal end of the duct, as there often is when the duct is completely severed, it is preferable to disregard it.

Stricture of the Common Duct with Immediate and Complete Retention of Bile. In case the duct has been severed and the proximal end closed, jaundice will immediately ensue and will gradually increase without pain, chills or fever. The content of serum bilirubin rises very rapidly. No bile is found in the duodenum or feces. The jaundice becomes very deep yellow and then changes to green. These patients require the most careful attention before anything can be done, and in spite of all precautions the risk of operation is very high. The longer the time since the jaundice became complete, the greater will be the risk. The amount of bilirubin in the blood serum is the best index to the degree of the risk, and no operation should be undertaken during the time the serum bilirubin is increasing. After prolonged preparation by increasing elimination, repeated intravenous

administration of calcium, and an occasional transfusion, certain of the cases of complete obstruction can be carried through an operation. Although calcium chlorid tends to increase the amount of serum bilirubin at first, its general effect is to decrease. If nothing is done to relieve the situation, it is surprising how long some of these patients continue to live under these conditions. Operating on this type of case is certain to be accompanied by a great amount of oozing as the adhesions are separated. These oozing surfaces should be controlled by ligature and suture rather than by gauze packs which invite a recurrence of the bleeding when they are removed. Transfusion should be given as soon as the operation is completed, even though it does not seem necessary at the time.

The technical steps in the operation can be carried out in a much more satisfactory manner by making the separation of the adhesions close to the surface of the liver. If the suspensory ligament can be identified, and the liver supported by traction on the ligament, it is possible sometimes to brush the adhesions away from the surface of the liver, and in this way expose the region of the severed duct without opening into the general peritoneal cavity. This is a great advantage when it can be accomplished and less reaction will follow this form of operation than that in which it is necessary to pack off the peritoneal cavity. As the surface of the liver is exposed and the region of the duct approached, a rounded projection which is tense and full of fluid is found where the hepatic duct formerly was. When this is opened clear or white fluid gushes out, as if it had been held under considerable tension. This fluid is the secretion from the glands in the wall of the duct and generally, as soon as it has escaped, bile-stained fluid appears. Anastomosis of this stump of duct to the duodenum is the best course to pursue. It is often impossible to tell which duct one is dealing with, and while it would be well to know whether it is the right or left hepatic or the common duct, still it is not essential. If one hepatic duct is intact and is anastomosed to the duodenum, that is sufficient. Damaged hepatic tissue regenerates very quickly, and if a large part of the liver is destroyed or removed, rapid compensatory hypertrophy occurs in the remaining portion.

The following case is one in which a complete stricture was found which seemed to be the result of trauma at the time of cholecystectomy:

Case IV.—A woman came to the clinic, December 3, 1925. Cholecystectomy had been performed in March, 1925, for cholecystitis with stones. She had suffered from typical attacks of gall-stone colic for three years previous to the operation, but jaundice had been invariably absent. At the time the gall-bladder was removed the surgeon stated that he had explored the common duct without opening it, and found it to be normal. The wound was drained. Three days after the operation the patient began to show some signs of jaundice. After a few days the wound opened and discharged a little bile. It opened and closed several times during the next eight weeks and, finally remained closed. As soon as bile stopped draining to the outside jaundice increased, pruritis appeared, the stools were clay-colored and the urine was dark. The patient had no pain during this time. Duodenal tubing indicated that a small amount of bile was passing through the duct, although jaundice was very deep. The serum bilirubin ranged from 8 to 12 mg. for each 100 cc. of blood.

About the middle of December, more than nine months after the cholecystectomy, and after considerable preoperative preparation (jaundice was very deep), an exploratory operation was performed. It revealed a stricture of the hepatic duct just below the surface of the liver. In this case the common duct was very readily exposed, so I opened it about 2.5 cm. below the surface of the liver, passed a large probe up into the hepatic duct and obtained a considerable quantity of bile. I dilated the stricture and put in a T tube, one limb of which extended well up into the intrahepatic duct. This seemed simpler than hepaticoduodenostomy, although if the trouble recurs after the tube is removed it may be necessary to anastomose the hepatic duct to the duodenum. This tube is to be left in from six to eight months.

Comment. This case illustrates those in which the symptoms come on immediately following cholecystectomy and persist until the secondary operation. It was impossible to ascertain at the time of this second operation just what might have occurred at the first one. In some cases the duct is undoubtedly severed, in others it may be clamped and ligated, while in still others it is possible that the reduction of the circulation to the tissues and the reaction in the tissues following the use of a considerable amount of drainage material might be factors in producing these strictures. Traumatic strictures will undoubtedly occur occasionally in spite of all precautions, and one method of removing the gall-bladder may be just as good as another. In order to avoid accident, one rule must always be followed, and that is, never to clamp or cut

what appears to be the cystic duct until it has been completely separated from all of the surrounding structures.

CONCLUSIONS. 1. Obliterative cholangitis resulting in stenosis of the common or hepatic duct is the cause of a considerable proportion of strictures of the common duct that have been classified as traumatic. These cases can often be recognized before the first operation. The symptoms are generally intermittent. Repeated operation is indicated if necessary.

2. Stricture of the common duct in which a biliary fistula exists may be deceptive in that there may be unexpected biliary cirrhosis. Careful study and any indicated preparatory treatment should be followed out in spite of the lack of jaundice.

3. Complete jaundice and severe biliary cirrhosis accompany the severance of the duct when the proximal end is closed. It is necessary to spend a great deal of time in preparing the patient for operation. It is also necessary to give much attention to the postoperative care.

4. Anastomosis of the stump of the common duct or the opening in the surface of the liver to an opening in the duodenum over a tube is the most satisfactory operation.

5. In certain cases recurrence of symptoms follows operation for stricture of the common duct. If these symptoms persist and increase further operation is indicated and with some prospects of permanent relief.

OPERATIVE MANAGEMENT OF COMMON-DUCT STONES

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Operations upon the common duct are still formidable and still carry much of their old risk. Stones in the common duct may be removed by a perfect technic, but the patient may die. The mortality of operations upon the common duct is as high as that of operations for the removal of tumor of the brain.

Because of the similar high mortality in these two groups of cases, I propose to offer evidence which tends to show that the disturbance of either of these two great organs by an operative procedure affects the organism in a similar manner. After operations upon either of these organs there may be a rapid loss of bodily energy; consciousness fades slowly, and in each case there is little, if any, medication which can influence the unfavorable course of the patient. The brain and the liver are alike highly sensitive to variations in temperature. Each is a powerful organ manifesting variations within it in a peculiarly dramatic way; each is essential to life.

In the case of the brain, through the development of an exquisitely protective technic, as is emphasized in the practice of Cushing, the forbiddingly high mortality of operations by untrained hands has been strikingly reduced. Are there corresponding possibilities in surgery which involves the innervation of the liver? It has been noted elsewhere that both the brain and the liver are master organs, whose functions are the result of chemical activity, and therefore are fundamentally affected by temperature variations. Each of these organs responds as a whole to every activity of the organism. Therefore, as one would expect, each has an extensive development of nerve supply. Each is sensitive to any disturbance of its nerve connections. In

view of this fact, it is strange to note that this primary essential to the maintenance of the optimum function of the liver has been little regarded, if at all, in the literature pertaining to this subject. This is perhaps due to the fact that interference with the nerve supply of this organ is not met by such immediately obvious and dramatic response as that which follows interference with nerve connections in the brain or the recurrent nerves, for example, in the course of a thyroidectomy. Let us, therefore, consider briefly the effect of temperature changes and of crude interference with the sympathetic nerves and ganglia of operations upon the common duct.

Sympathetic Nerves and Ganglia. Within the field of operations upon the common duct lies what might be termed the "brain" of the sympathetic nervous system—an exquisitely sensitive structure which is the center of a tangled network of extremely sensitive sympathetic nerves which supply the organs whose function is essential to life, among them the liver. Moreover, a network of fibers from the sympathetic nerves and from the left vagus nerve surround every bloodvessel and capillary and every duct within the liver. By means of this extensive nerve supply the liver, like the brain, is completely coördinated with the organism as a whole. Because of the inter-relations with the other viscera through the sympathetic ganglia, from which the principle nerve supply of the liver is derived, in operations upon the common duct the surgeon may greatly interfere not only with the innervation of the liver but with the innervation of the pancreas, of the adrenal glands, of the stomach, of the intestines; indeed, every part supplied by the sympathetic nervous system may be affected. Obviously, in the course of a vigorously aggressive operation upon the common duct the surgeon is doing to the abdominal brain what would correspond to the effect of a digital search with a crude finger throughout the real brain. Thus a crude search for a common duct stone would correspond with a crude search for a tumor in the brain. This line of reasoning is pertinent only if it can be proved that any mechanical disturbance of the sympathetic ganglia, of the fibers of the sympathetic nerves, or of the vagus does produce a gross disturbance of bodily function, in extreme cases leading to death. In support of this reasoning, therefore, the following facts may be presented: (1) The clinical effects of a breaking-up of dense adhesions in the

course of a search for the common duct causes a general bodily effect comparable to that produced by manipulation of the spinal cord or of the brain. (2) Experimental trauma of the sympathetic nervous system—interference with the sympathetic nerve supply—produces cytologic changes in the liver cells as well as in the brain cells. (3) Strong emotional stimuli cause cytologic changes in the cells of the liver. (4) Blocking the splanchnic nerve supply greatly reduces the systemic effects of manipulation of the viscera.

The point to be emphasized is that not only is the function of the liver interfered with by rough manipulation of the common-duct region, but that the effect is body wide. Clinical experience and experimental researches make it clear that because of the above considerations; operations upon the common duct require a wide regional block with novocain and, when feasible, a splanchnic block; a clear exposure; a sharp, feather-edge dissection, as has been so clearly demonstrated by Deaver; a bloodless field, a suction apparatus being used to pick up bile or any oozing of blood. Such a technic meets the factor of nerve injury in common-duct operations extremely well. The details of the technic for searching for a stone in the common duct need not be repeated here.

Up to the present point we have been considering only such injury as may be inflicted during the performance of the operation itself. Nerve injury may be produced, however, after the operation by drainage. There is no excuse excepting absolute necessity for the introduction of a drain of any kind into the field of the sympathetic ganglia and sympathetic nerve fibers when a better drain can be placed in the right flank, terminating in Morrison's pouch.

Another factor of danger is the drainage of the common duct itself. Sudden decompression of bile in a jaundiced patient may have the same effect on liver function as the sudden decompression of urine in the case of obstruction of the urinary system. The normally closed biliary system maintains adequate bile pressure within the system; therefore, instead of draining the common duct, I close it just as I would close incised intestines. Normal pressure of bile is considerable, and presumably has functional importance. Drainage of the common duct under any circumstances reduces this pressure to zero; if there is a sudden release

after a high back pressure due to obstruction the effect is the same as that produced by the sudden breaking of a dam, *i. e.*, the pressure is temporarily reduced far below the normal level. It should be borne in mind also, in connection with the question of drainage, that faulty drainage may produce a well instead of a stream. Too much fluid may accumulate before it escapes, with immediate resultant functional disturbance and the more remote result of adhesions, which in turn interfere with liver function and, consequently, with general organic function. This damage is met or largely eliminated by employing gravity drainage through Morrison's pouch. Owing to the position of this pouch, the accumulation of fluids will be avoided, and the resultant adhesions will not be at such vulnerable points as those encountered by an anterior drain. When this method of drainage is employed the primary abdominal incision can be closed at once. When the common duct is thus closed and the drain is inserted laterally in Morrison's pouch, as there is no drainage in the common duct there will be less scar tissue and fewer adhesions in this vulnerable field, and both the immediate postoperative convalescence and the clinical end-results will be by so much more improved.

By the technic thus described the operation has been carried along with the minimum anesthesia, with minimum disturbance of the sympathetic nervous system and, in consequence, minimum disturbance of the vital organs. But another factor remains to be considered. Since the liver is the greatest chemical factory in the body, since it furnishes one-third of the heat of the body, since the liver is essential to life and its activity will balance with that of the brain, and since these activities depend on chemical activity, and chemical activity is influenced by temperature, the relation of the temperature of the liver to operations which impinge upon it remains to be considered.

By experimental researches in the biophysical laboratories of the Cleveland Clinic it has been found that the introduction of heat within the abdomen causes an immediate rise, not only in the temperature of the liver but also in the temperature of the brain, the rise in the temperature of the brain occurring synchronously with the change in the temperature of the liver. It would follow from this observation that the application of heat to the liver should in large part counteract the effect of operations

upon the liver and bile ducts. As a result of our interpretation of this experimental fact, we have been applying heat to the liver by means of diathermy, which we have found to be an ideal method of holding the temperature of the liver at a normal level. One plate of the diathermy apparatus is placed on the lower chest on one side and the other is brought opposite the dome of the liver. The current can thus be continually applied during the operation, and the temperature of the liver and the abdominal viscera can be maintained at or above the normal throughout the operation regardless of the exposure of the intestines; moreover, the application of the diathermy current during the immediate postoperative hours is of great aid in carrying the patient through that critical period.

In our present series of cases of common-duct stones—still small—which have been managed according to the plan described above our results have been strikingly in contrast with the results of operations before this plan was adopted. Under this plan the mortality and morbidity attending operations upon the common duct should show a like change to that which has followed the present plan of management of operations upon the brain and of operations for hyperthyroidism.

		Cases.
Total number of gall-bladder operations (April 17, 1926)		1682
Cholecystectomy	740	
Cholecystotomy	786	
Total number of operations on common duct		83
Before 1921, operative mortality	11.6 per cent	
1921 to 1926 (35 operations), operative mortality	5.7 per cent	

STRICTURES AND OPERATIVE INJURIES OF THE BILE DUCTS

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Operative injuries to the bile ducts, while not frequent are met with sufficiently often to make a study of the reason for their occurrence and the best method of repair of some interest. The reports in the literature are usually of one case or a limited series of cases, which are reported in order to demonstrate some method of repair. Considerable series of cases showing the end-results of the various methods of repair have been seldom reported.

Cause of Injury. The operative mortality, the frequent necessity of reoperation and the frequency of recurring symptoms make it worth while to consider the causes of injury and the method of avoidance It is of interest that the accident has occurred at the hands of the best surgeons, sometimes in an easy case, and it may result either when the operation is started at the cystic duct and the cholecystectomy done from below upward or from the fundus downward. But the accident has occurred here usually because of too much traction in the effort to remove the whole cystic duct, which it is better not to attempt, as injury to the common hepatic duct is more likely to occur than stones to form in the small amount of cystic duct remaining.

In the more difficult cases there may be: 1. Insufficient exposure, causing failure to recognize the cystic and hepatic ducts.

2. Traction on the gall-bladder, causing angulation, with clamping of a part or the whole hepatic duct.

3. Bleeding from an unseen point, resulting in a blind effort to clamp an invisible vessel, so that the duct is clamped, the clamp being left *in situ*, or the duct tied off around the clamp by means of a suture on a needle.

4. Inaccessibility of the duct may be due to a distended, inflamed and thickened gall-bladder, which causes marked shortening of the cystic duct.

Anatomy and Congenital Abnormalities. In addition to these causes, in which the normal anatomy of the parts may be changed by pathologic conditions, there are other conditions increasing the danger of injury, due to congenital anatomic abnormalities. Eisendrath describes these abnormalities, which may occur in the ducts and the vessels as follows:

1. The cystic duct does not always unite with the hepatic at an acute angle, as occurs in 75 per cent of the cases. In 17 per cent it may run parallel with the hepatic duct, and its terminal 2 cm. or more may be united to the hepatic duct by fibrous tissue.

2. In 8 per cent the cystic duct makes a twist in front of or behind the hepatic duct.

3. More rarely there may be accessory hepatic ducts, which may empty into the common or cystic duct or into the gall-bladder or there may be a double common duct.

There are an equal number of possibilities of variations in the bloodvessels The cystic artery may be double in 12 per cent of the cases and does not always arise from the right hepatic. It may arise from the gastroduodenal artery, a branch of which crosses in front of the common duct in 76 per cent of the cases. The right hepatic artery lies behind the hepatic duct in 70 per cent of the cases, and the cystic artery arises just beyond the right edge of the duct. But in 12 per cent of the cases the right hepatic artery passes in front of the right hepatic duct, and in 10 per cent runs parallel to the cystic duct, close to the neck of the gall-bladder, before entering the right lobe of the liver. In 8 per cent of the cases the right hepatic artery crosses the right edge of the main hepatic duct and then enters the liver, or forms a ring around the right hepatic duct

While these anatomic abnormalities are to be kept in mind, the fact remains that the accident by far most frequently occurs in the absence of anatomic abnormalities rather than as a result of this cause.

Site of Injury. The location of the injury is frequently described as of the common duct, but as a matter of fact it usually occurs where the cystic and hepatic ducts join or above this point. When the accident results from traction on the cystic duct the

resulting injury occurs at the point of union of the three ducts. When traction causes angulation, and the duct is cut, it is at the position where the cystic and hepatic ducts unite to form the common duct or higher up in the hepatic. When a clamp is applied without proper visualization of the duct, in an effort to stop bleeding, it is frequently on the hepatic duct itself. In many instances, at operation, it is found that the whole main hepatic duct up to the bifurcation in the transverse fissure of the liver is missing.

The point of union of the cystic, hepatic and common ducts is the narrowest part of the common duct, and the custom occasional employed of draining the common duct by inserting a tube at the point of mergence of the cystic duct may result in subsequent stricture by ulceration or destruction of the duct, a large portion of which may slough out in the presence of severe infection with cholangitis. This occurred in one of my own cases (Case II).

Pathologic Condition. There may be a discharging sinus, by which all the bile is discharged on the abdominal wall; or a patient with no sinus who is suffering from either a deep jaundice, when the obstruction is complete, or has a history of intermittent attacks of jaundice, pain and temperature elevation, due to a recurring cholangitis. The stricture may be only a narow band (Case VII), or it may extend over a distance of 2 cm. or more (Case VIII).

At operation the area beneath the liver in the region of the transverse fissure and about the common duct is occupied by a mass of adhesions. It may be possible to find both ends of the duct. The lower portion may be found only with great difficulty in these adhesions, or may not be identified. In many cases the stump of the hepatic duct, or the right and left hepatic ducts, may be retracted into the transverse fissure of the liver and appear as a small, fluctuating area which can only be identified by aspiration of bile with a fine needle. In some cases, instead of forming a common pouch, they may be divided, each duct being closed off separately (Case I). Frequently a stricture remains patent, but very narrow, and it closes when cholangitis develops. Thick, inspissated bile or sandy material or even small stones may form behind and cause blocking of the stricture (Cases VI and IX).

It is stated that, in reconstructed ducts, epithelium forms rapidly to line the reconstructed area. While this has been proven experimentally on animals, it is still doubtful how much epithelium forms along the line of a reconstructed duct which has been repaired after an injury, particularly in the presence of cholangitis and infection. It would also appear to me that even if epithelium did form, as long as there has been any infection present, the surrounding scar tissue is going to contract and cause subsequent narrowing in many of these cases. In an autopsy protocol of one case reported in this article (Case VIII), in which the stricture extended for over 2 cm., there were isolated spots of epithelium, shown by the microscope, along the strictured area, but most of the strictured area contained no lining epithelium.

When the obstruction is almost complete the liver is enlarged, with dilated intrahepatic ducts, often filled with thin, pale, watery bile. If the obstruction is relieved at operation by opening the hepatic duct, or if the liver is cut into at autopsy, these dilated ducts collapse with a marked dimunition in size of the liver. There is a varying amount of cirrhosis, apparently related to the amount and length of time the cholangitis has existed, and the liver may be hard and contracted, with a cirrhotic spleen and portal obstruction (Case VIII). Microscopic examination of the remaining duct shows round-cell and leukocytic infiltration as evidence of infection.

Symptoms. When due to operative injury the symptoms usually, but not always, follow a postoperative history of prolonged drainage of bile from the wound. This drainage finally ceases or a permanent fistula remains. When drainage ceases the appearance of symptoms varies as to time: Occasionally several years may elapse; then occur attacks of jaundice, pain, usually temperature elevation and clay-colored stools. This may become progressively worse or the patient may continue to have attacks of varying severity over months or years, finally requiring operation. The symptoms will vary, depending on the degree of obstruction and amount of cholangitis or the combination of both. It is sometimes difficult to reconcile the appearance of symptoms with the pathology found at operation (as in Case I). In this patient there was no biliary discharge following operation, the wound healed by primary union, and jaundice and signs of obstruction did not appear until four months later, although at operation the whole

main hepatic duct was missing up to the bifurcation in the transverse fissure of the liver and the common duct was separated from this by almost 2 cm. It would appear that during this four-month interval there must have been some passage between the open divided ends which finally closed.

Methods of Repair. There are many methods of repair suggested 1. An end-to-end suture of the damaged duct, either with or without drainage, by means of the introduction of a T-tube; a tube through the ampulla of Vater; or with a tube leading through the stricture and coming out through an opening below in the common duct, and then led through the abdominal wall.

2. A division or excision of the stricture, with the use of a tube in any of the previously mentioned methods.

3. Repair over a tube, with an attempt to use the round ligament, the liver, the intestine, the neighboring peritoneum or the omentum to replace the missing portion of the duct.

4. Hepaticoduodenostomy, with the implantation of the remains of the cystic duct into the duodenum or stomach over a tube, more rarely into the jejunum or even into a segregated loop of small intestine. (Kauch.)

5. Hepaticoduodenostomy, with an attempt at partial reconstruction of the duct out of a flap made from the duodenal wall, or suture of the lower edge of an opening in the duodenum to part of the stump of the duct, the upper edge being sutured to the liver capsule. (Mayo.)

6. The implantation of the sinus into the duodenum or stomach.

In addition to these methods, cases have been reported of a lateral anastomosis of the duct to the duodenum. Cholecystenterostomy or cholecystgastrostomy has been done where the stricture has occurred and the gall-bladder has not been removed and is still functioning. Also the injury in the common or hepatic duct has in some instances been repaired by using a graft from the remains of the cystic duct (Kehr, also Judd) or the gall-bladder wall, and by telescoping the injured hepatic into the stump of the cystic. (Walters.) Cases are also reported of attempted repair by facial flaps, and such desperate measures, when no duct could be found, as hepatostomy, cholangiostomy and hepato-enterostomy.

A choice of which of these operations should be employed would necessarily depend in many instances upon the condition

met with; but where such choice may be voluntary, it would be of assistance to determine if possible the end-result of the various types of operation. An analysis of 12 cases from the Surgical Service of St. Luke's Hospital, together with a report of some of the other series of cases published, is the basis of this study. Many of these patients have had repeated operation; several have been reported as cured or well, only to have a recurrence of their symptoms, sometimes after as long as two years of being symptom-free, and have died as a direct result of their condition, which would make questionable the statement publishd in the *Annals of Surgery* in 1924, which says: "Patients who recover from the operation usually have mild attacks of colic, with or without jaundice, for several months or sometimes several years, and then completely recover."

Case Histories

Case I.—S. T., female, aged thirty-two years. Cholecystectomy in another hospital, June, 1924. Had temperature following operation. Wound healed by primary union. Symptoms of jaundice and cholangitis in November, four months later. Bile index 167. At operation, December 30, 1924, a stricture was found at the junction of hepatic ducts with a separation of between 1 and 2 cm. of stricture from the upper end of the normal duct below. Reconstruction by end-to-end suture over a T-tube in December, 1924. The patient was well five months and then had a recurrence of symptoms which lasted until November, 1925. She had no symptoms since that time and gained 15 pounds in weight. Bile index at the present time 9. She is now well, eighteen months after operation and symptom-free for six months. (Douglas.)

Case II.—D. H., female, aged fifty-four years. Cholecystectomy in October, 1919. Gangrenous gall-bladder, stones in the common duct, cholangitis (bad infection). Part of the duct sloughed out with the drainage tube which had been sewed into the wall of the duct. Symptoms appeared August, 1920, eight months after operation—jaundice and cholangitis. A second operation was done October, 1921, two years after the previous operation. Closure of the ducts at bifurcation, which was connected with the common duct by a strand of tissue representing the posterior wall of the duct. Reconstruction by suture over T-tube. The patient well one year, then symptoms recurred and persisted until May, 1923. She has been well since then (four years) and has gained 73 pounds in weight. (Douglas.)

CASE III.—L. H., male, aged forty-six years. Cholecystectomy in another hospital, June, 1919. Sinus never closed. Profuse bile drainage. Operation, December, 1919. Ducts found cut at the junction of the hepatic with the common duct. The ends of the cut ducts were sutured together. There was profuse drainage for eleven days which stopped on the thirteenth day. The patient is now well after six years. (Downes.)

CASE IV.—E. H., male, aged forty-four years. Cholecystectomy in another hospital in 1917. Clamps were left on for hemorrhage for forty-eight hours. He had attacks of jaundice for six months after the operation and was well for three and a half years. For the next year he had nine or ten attacks of pain, colic and jaundice. At operation, June, 1922, a stricture was found at the hepatic bifurcation $\frac{1}{8}$ inch in length which would not admit of a probe. The hepatics were much dilated. The stricture was cut, a T-tube inserted and left in one month. Two months later there was a recurrence of symptoms. A third operation was done in October, 1922. Recurrence of stricture which was again divided and a T-tube inserted. Well since (four years). (Downes.)

CASE V.—J. V., female, aged twenty-two years. Cholecystectomy in another hospital in August, 1919, followed by chills, fever and persistent sinus. Second operation was done in another hospital in November, 1919, for persistent sinus. This was unsuccessful. Operation in February, 1920. Stricture found in right hepatic duct which had been divided and retracted into liver. The left hepatic duct was apparently continuous with the common duct. No effort was made to suture the duct at this operation. The fistula failed to heal and reoperation done August, 1920. The duct was sutured over tube and led out through the common duct above the duodenum and out through the abdominal wall. She has now been well six years. (Mathews.)

CASE VI.—G. S., female, aged fifty-four years. Cholecystectomy in another hospital in 1922. She was in the hospital for six months, then returned home, but had attacks of pain at times and continuous jaundice. A second operation for stone in the common duct was done in 1923, when she was in the hospital for six weeks, after which she was well for eight months, then again had attacks of pain and almost continuous jaundice. Operation, April, 1925. The patient was jaundiced. A dilated hepatic duct was found which was opened and drained. A probe could be passed up to the liver, but not down into the common duct. Reoperation April 28, 1925. Sinus running down to dilated

hepatic duct. Common duct not found in adhesions. Hepaticoduodenostomy over tube. Tube remained in place over two months and patient remained well until January, 1926 (nine months) when the symptoms returned and she was again operated on in February, 1926. At this time the duodenum was opened, the point of anastomosis of duct and duodenum dilated and inspissated bile and mucus escaped through the opening. The tube was inserted into the duct and down into the duodenum and the duodenum closed. The wound healed and the patient was discharged three weeks later. She again returned to the hospital six weeks later with some symptoms of jaundice and cholangitis, which cleared up in a few days. This was only two months ago—end-result cannot be stated. (Downes.)

Case VII.—S. S., male, aged forty-two years. (Case reported in *Annals of Surgery*, 1918, lxvii, 619.) Cholecystectomy in December, 1917. The hepatic duct was cut at its junction with the cystic. Immediate end-to-end suture was done. The bile discharged for six weeks and then stopped suddenly. The patient well until September, 1919 (twenty-eight months) then had attacks of pain with chills and some jaundice to January, 1923, when he was again operated on. The stricture had recurred which was divided and a T-tube inserted. The tube continued to discharge bile and was left in for seven months. The stools showed bile and the eyes were jaundiced at times. The biliary fistula persisted. The patient was readmitted to the hospital, June, 1924 and died of mesenteric thrombosis. Autopsy showed a fistula draining the common duct. Hepaticus above the stricture was dilated and contained inspissated bile. There was a firm scar tissue band at the center of the duct. The lumen was small but patent. Cirrhosis of the liver. (Downes.)

Case VIII.—L. H., female, aged forty-eight years. Cholecystectomy in another hospital in 1916, followed one month later by jaundice. At operation in same hospital the common duct was not found and hepaticoduodenostomy over tube was said to have been done with an attempt to reconstruct the lower end of the duct from the duodenum. The tube was in place for several months. Since then she has had intermittent attacks of pain, temperature and jaundice. Operation September, 1917, in St. Luke's Hospital. A stone was found impacted in the ampulla of Vater and removed transduodenally. Her condition improved, most of the bile entering the duodenum, but she had nausea, temperature and slight jaundice at times. Reoperation, November, 1917, when the hepatic duct was drained for cholangitis. It contained inspissated bile. Hepaticoduodenostomy not interfered with. The

drainage ceased and the patient was in good health, gaining 40 pounds, until June, 1919, when she again had chills, fever and jaundice when the duct at the hepatic fissure was again opened and drained. At this time there was bile in the duodenum, showing that the obstruction was not complete. The patient died in October, 1925 with ascites and deepening jaundice. Autopsy showed cirrhosis of liver and a large spleen. The ducts were only moderately dilated. Beginning at the portal fissure there was an irregular strictured portion of duct at least 1 inch in length and about the caliber of a knitting needle. Below this, the duct appeared normal. It was the opinion of the operator, Dr. Mathews, that a hepaticoduodenostomy had not been done, as stated, at the previous operation because of the continuity of the common duct, but that the anastomosis must have been made with the cystic instead of the hepatic duct. (Mathews.)

Case IX.—E. K., female, aged thirty-nine years. Cholecystectomy in 1914. A part of the common duct was excised with immediate suture and repair over a tube into the duodenum. A permanent fistula developed. Six months later there was a reoperation when a stricture $\frac{1}{2}$ to $\frac{3}{4}$ inch was found. The stricture was excised and the posterior wall sutured, but the anterior wall could not be approximated. The defect was covered anteriorly with round ligament and omentum. The patient was well two and a half years then reoperated on by another surgeon in October, 1916, who removed biliary calculi from above the stricture and dilated the latter. In January, 1917, the symptoms recurred and operation was done by a third surgeon and resulted in the death of the patient. (Lyle.)

Case X.—O. O., female, aged twenty-nine years. Cholecystectomy in another hospital in 1916 which was drained for five months and was followed by fever, chills and jaundice. She was operated on in St. Luke's Hospital in May, 1918. There were many adhesions. The common duct was not found. There were many glands in region of the ducts. Bile was finally obtained from the hepatic fissure of the liver and was drained. The patient died after three blood transfusions. Autopsy showed hepatic ducts dilated to the size of the ring finger and completely closed off apparently from the remains of the common duct by adhesions and lymph nodes, but probably some small lumen was present, as some bile had been present in the stools during the past year. (Mathews.)

Case XI.—R. P., female, aged twenty-three years. Cholecystectomy from below in November, 1924, which was followed by profuse biliary drainage. The patient was discharged one month after the

operation with the wound healed and bile in the stools, but returned in two months with jaundice. She was pregnant at this time. She went through the pregnancy and had five or six attacks of pain when the jaundice would get worse. At operation November, 1925, the common duct was not found, the liver was enlarged and contained several cystic masses. In the portal fissure, a dilated hepatic duct was found containing white bile. Tube drainage of the hepatic duct. The patient died five days later from hemorrhage after three blood transfusions. (Downes.)

Case XII. –V. S., female, aged forty-eight years. Cholecystectomy for cholelithiasis, December, 1924. The bleeding following the removal of the gall-bladder was stopped by clamp and suture ligature. The operation was followed by biliary fistula and acholic stools. The patient refused reoperation but was reoperated on several months later abroad and died as a result of the operation. (Westerman.)

Analysis of these 12 cases shows that 5 are well and symptom-free from one and a half to six years after operation (Cases I, II, III, IV, V). Of these, 2 had second operations (Cases IV and V). All were cases of suture of the ducts: 3 over a T-tube (Cases I, II, IV); 1 with no tube drainage (Case III) was well six years; 1 with tube led out through the common duct below the point of repair (Case V) was well six years. Two had reappearance of symptoms after operation which lasted four to twelve months, but are now symptom-free eighteen months to four and a half years after operation (Cases I and II). One patient had a hepaticoduodenostomy and was well nine months; recurrence due to narrowing of the lumen and obstruction from inspissated bile; reoperation and return of symptoms two months later (Case VI). Too recent to give end-result.

One patient was well almost two years after immediate end-to-end suture, then reoperated, the stricture cut and a T-tube inserted. The sinus never closed, the symptoms occurred at intervals, and the patient died one and a half years later of mesenteric thrombosis (Case VI). One died after four operations during eight years (Case VIII). One after repair over tube into duodenum, six months following immediate suture repair was well two and a half years, then had a third operation, in another hospital, from which she died (Case IX). Two died of drainage operations, both of secondary hemorrhage (Cases X and XI). One of a repair in another hospital (Case XII).

It is obvious that no justifiable conclusions could be based on this series alone Study of other series are of interest. Judd reports 10 cases. Of these, 4 died (2 as an immediate result of operation), 1 two years and 1 four months after hepaticoduodenostomy (Cases I, III, IV, V). Three cases were well and symptom-free. One in which stricture was divided and T-tube used was well seven and a half years (Case II). One repair of defect by insertion of a piece of the cystic duct, well seven and a half years (Case VI). One of transverse suture after splitting stricture and T-tube used, was well three and a half years after two years of symptoms (Case VII). Three cases still complained of some symptoms: Case VIII had three operations—division of stricture with T-tube, a lateral anastomosis and a hepaticoduodenostomy—was in good health for three years but at times jaundiced; Case IX had two operations, in both the stricture was cut and a T-tube inserted—was in good health for two years but has had recurrence of pain; Case X had two operations, both hepaticoduodenostomies one year apart, and one and a half years later had attacks of pain and jaundice.

Seven cases of hepaticoduodenostomy are reported by Erdmann, which are classed as 8 operations with 7 patients. Two died (Cases III and V). Case I was well five years after second operation (hepaticoduodenostomy). Case IV, was well five years after the operation. Case VI had two operations and not reported cured. Case VII, tube in place eleven months—reported well three months later. Case VIII, reoperation five months after hepaticoduodenostomy, and only reported as convalescent.

McArthur has reported 5 cases of repair of the ducts over a tube into the duodenum. The point of importance in these cases being the use of a cuffed tube or the funnel end of the tube which was placed in the duct above the stricture, a long portion of the tube going into the duodenum. A personal communication recently received from Dr. McArthur gives the final report in these cases. Case I died two years after operation from carcinoma of the stomach. Case II died one and a half years after operation from septic cholangitis from the presence of a short tube that never passed, as described in his paper. Case III is still alive and free from jaundice eight years after operation, but every month or so has an attack of chills, fever and vomiting, suggesting intermittent cholangitis. Patient had a gastroenterostomy five years

ago for pyloric stenosis. Case IV is still well. Case V was operated on in 1913 by Dr. Finney, who found stone in the common duct which contained a fragment of rubber, probably from small portion of catheter. Believes patient is still alive.

In the series of 51 cases from the literature published by Eisendrath in 1920 and including cases of duct injury from the series of Eliot, reported in 1918, there were 51 cases of injury to the bile ducts. Of these the result after one year is only reported in 4 cases. The rest, in which any time is mentioned, are reported as dead as a result of operation, or recovered One is reported as against having symptoms. There are 5 cases of operative injury with immediate transverse suture of the hepatic duct over a T-tube, quoted from Kehr's *Surgery of the Bile Passages*, with the report, "all recovered and remained permanently well," but with no detail as to time mentioned. Of the cases reported as well over one year in this series, 1 by McArthur died two years later of carcinoma of the stomach; and 1 by Lyle (Case IX) died later as a result of operation for recurrence of symptoms. Two cases in this series are of especial interest. One reported by Wolf, in which the gall-bladder was anastomosed to the common duct, was reported well four years later; and 1 reported by Jenckel, in which there was an obliteration of the common and lower part of the hepatic ducts for a distance of 8 cm., which was repaired by means of a tube from the hepatic duct into the duodenum. The patient remained well four years after operation, and the operator states that four weeks after operation, when the tube was removed, the canal, lined by epithelium, was found. Of the remaining cases it is of interest to state that 1 case, reported by Downes in 1918 as a case of recovery, died in 1924, having been reoperated with recurrence of symptoms; and also that the case in this series reported by Stettin, of immediate suture following the injury in 1915, was perfectly well at the time of the last report, in 1923, nearly nine years after the operation. Two of the cases in the series are those reported by Erdmann, and the later results are found in his report, here abstracted. The literature contains many reports of isolated cases, in addition to this series, but as end-results are not given they can add little to the information here sought.

Mention should be made of the method of treatment where a biliary sinus still persists by means of implantation of the sinus into the stomach or duodenum—this was unsuccessfully attempted

by von Stubenrauch in 1906. One of the first cases in which this was temporarily successful was by Murphy, who implanted the sinus into the common duct below the stricture, the patient dying eight months later with recurrence of symptoms. Several other attempts by this method have failed, but Lahey in 1924 reported 3 cases—1 done two years previously and remaining well; the second patient died of carcinoma of the pancreas a few weeks later. In both of these the sinus was anastomosed into the duodenum. The third case, in which the sinus was anastomosed into the stomach, had been done eleven months previous to the report, which stated that the patient was now well, but had twice had attacks of jaundice accompanied by chills. Lilienthal has also reported on a similar operation, done in December, 1921, in which the fistula was dissected out and implanted in the stomach near the pylorus, and a report from her family physician states that she has been perfectly well since then—four years later. St. John, at a recent meeting of the New York Surgical Society, presented a case in which similar operation was done, the patient being symptom-free twenty-one months after operation. If this method is employed the sinus should not be dissected down below the margin of the liver, to the lower surface of which it is usually attached, because of danger of injury to the sinus wall in attempting its separation.

Examination of the end-results of the series of 12 cases followed at St. Luke's Hospital, together with the reports of the other series of cases, makes any definite conclusion as to the best method of repair difficult if not impossible. Certain conditions limit the choice of operation, such as the location of the injury, the amount of destruction of the ducts, difficulty in approximating the divided ends, the amount and density of the adhesions, the presence or absence of a sinus, the condition of the patient, such as the degree of jaundice, the amount of impaired liver function, or actual destruction of or change in the liver tissue. It has been stated by previous writers on the subject that the two main factors of recurrence of symptoms are cicatricial contraction and ascending infection, causing cholangitis. In various methods of repair by means of hepaticoduodenostomy or implantation of a sinus, attention has been called to the absence of the sphincter of Oddi as the cause of this ascending infection; but in the St. Luke's series of cases, in which suture of the duct was done, cholangitis,

as shown by jaundice, chills and fever, was not of infrequent later occurrence (Cases I and II). It would appear from the St. Luke's cases, as well as those of Judd's, that the cases of direct repair without a tube or with a T-tube were the ones that gave the best end-results in the largest number of cases, although several of these had recurrence and required reoperation; while those in which a repair was done and a deficiency left for some other tissue to bridge over the gap, in many instances had recurrence of symptoms. Sullivan's case, however, was reported well four years after operation. Successful bridging over a long gap is not impossible, as shown by the cases of Jenckel, Kehr and Riggs, who were able to mobilize the ends of the duct and perform an end-to-end suture after a separation of 2 to 3 cm. The patient of Riggs being reported well four and a half years later.

Immediate recovery, as shown by McArthur's series, is usual in the cases of tube reconstruction The end-result over a long period of any number of similar cases cannot be determined from the data collected. The same statement must be made as to the results of hepaticoduodenostomy. One patient on which this was done in the St. Luke's series has required subsequent operation and has had recurrence of symptoms since then. Of the Erdmann series, only 2 were reported well for a period of five years. Of Judd's series, 2 cases had attacks of jaundice at times after hepaticoduodenostomy. There are, however, several cases in the series collected in the excellent article by Eliot on "Repair of the Hepatic and Common Bile Duct," published in 1918 and not included in Eisendrath's report, which show a successful result of implantation operations for a period of years—1 of Dujarier's, well three years, and 1 of O'Day's, well six years, after implantation of the duct into the stomach. And successful cases of hepaticoduodenostomy —well seven years, reported by Summers; four years, by Bazy; four years, by Mann; three years, by Crile; and fifteen months, by Wilms—are included in this collection of cases; also the case of W. J. Mayo, of implantation by his method, well more than fifteen years.

Reoperation or autopsy on patients who have had recurrence of symptoms, or died after their operation, shows that frequently there is a canal which remains patent, although this canal is usually markedly narrow in caliber, and that the attacks of cholangitis and jaundice are due to the obstruction caused by inspissated bile

or the formation of small stones, or detritus behind the point of obstruction. In 2 of the cases of the St. Luke's series (Cases I and II) this occurred after operation, and after a period of from four to nine months in each case, with the administration of large doses of bile salts, these symptoms cleared up and the patients have remained well since—1, a year and a half after operation and 1 almost four years. Whether this was due to the medication or not, cannot be definitely stated. It is not, however, illogical to suppose that the condition which favored the formation of stones, for which the original cholecystectomy resulting in the duct injury was done, may persist, and be favored by a partial obstruction or stasis in the duct. Therefore, anything which would help to keep the cholesterin in solution and favor the increased flow of bile may be of benefit, if symptoms of partial obstruction or cholangitis appear.

If any conclusions can be arrived at from the data studied, it would appear, that when possible to mobilize and approximate the ends of the ducts, direct suture is the method of choice, although here, too, occur some recurrences. Perhaps the more favorable results are partly due to the lesser amount as well as of density of adhesions that makes this procedure possible. If a tube is used in this operation, it seems of importance not to choose one that has too large a caliber, as pressure may cause necrosis or destruction of the epithelial lining of the duct. If direct union cannot be accomplished, hepaticoduodenostomy, either over a tube or by the direct method of Mayo, when the duct is very short and the duodenum can be mobilized, seems to be the second choice; while the method of reconstruction of the duct over a tube becomes a last resort when other methods are impossible. Occasionally, methods of repair of a defect by means of a portion of remaining cystic duct or implantation of the remains of a cystic duct into the hepatic may be employed, but the opportunity to use such a method would only be in the occasional case. The same circumstances apply to the implantation of a persistent sinus. If the patient is deeply jaundiced and in poor condition, and mechanical conditions make further operation hazardous, it is frequently advisable to drain the hepatic duct above the obstruction and defer the repair operation.

Whatever operation is done, there is a considerable percentage of recurrence. In a certain number of cases there may be a return

of symptoms, either of obstruction or cholangitis or both, for a number of months or years, which then clear up and the patient remain well. Many patients, however, in whom these symptoms intermittently recur will become worse and require reoperation. Others may remain well for months or even years and then have all the evidence of a recurring stricture.

SUMMARY. 1. Injuries to the bile ducts may occur in a simple operation or be due to pathologic conditions rendering the operation difficult, or to congenital abnormalities of the ducts or arteries. Traction on the cystic duct, lack of visualization or blind attempts to stop hemorrhage are the most common causes.

2. The site of the injury is usually at the point of union of the cystic and hepatic ducts or the main hepatic duct above this point; less frequently of the common duct.

3. The symptoms are those of biliary obstruction, with or without cholangitis, usually intermittent in character, the obstruction later becoming permanent; more rarely a persistant biliary fistula is present.

4. The pathologic condition present may show a narrow caliber stricture, which is very short to 2 cm. or more in length, above which small stones, mucus or biliary detritus is often found. The ducts above the stricture are dilated and the liver enlarged and soft, or may be cirrhotic when attacks of cholangitis have persisted.

5. Methods of repair are numerous and must depend on the condition found. Recurrence of symptoms are reported after all methods. Examination of follow-up reports appear to indicate that the best end-results follow suture of the ducts when possible. The next most favorable results, where a number of cases are reported, follow hepaticoduodenostomy.

6. Recurrence of symptoms may occur after the patient has been apparently well for months or years, or symptoms may disappear after several months of recurrence.

7. In two cases reported, the disappearance of late symptoms seemed to be influenced by the administration of bile salts.

8. Report of 12 cases from the Surgical Service of St. Luke's Hospital of New York is given.

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SOME NEW PHASES OF THE PHYSIOLOGY OF THE BILIARY TRACT*†

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The gall-bladder is known to have two functions, of which one is concerned with the regulation of pressure within the biliary tract, and the other is concerned with the concentration of bile by the absorption of water. Possibly one other function is suggested by the fact that the blood from the gall-bladder empties into the portal vein. It might be inferred from this fact that perhaps something is formed in the gall-bladder which is necessary for the liver to use. At the present time nothing is known about this possible additional function, although Sweet[1] has produced evidence which suggests that the gall-bladder forms something which acts to deësterize cholesterol esters.

Curiously enough, even the question of how the gall-bladder empties itself has aroused interest only within recent years. In 1917, Meltzer[2] proposed his now-famous hypothesis of the existence of a contrary innervation between the sphincter of Oddi and the gall-bladder whereby a dilatation of the sphincter was supposed to be accompanied by an active contraction of the gall-bladder. It is upon this hypothesis that Lyon devised his method of draining the gall-bladder by means of the instillation of magnesium sulphate into the duodenum. Sweet was perhaps the first one to raise

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serious questions against the validity of the hypothesis. He called attention to the anatomical difficulties which might act as obstacles against the emptying of the gall-bladder through the cystic duct, such as the acute angle which the cystic duct usually makes with the common duct, the presence of the Heisterian valves, etc. There is now, however, a large amount of evidence which seems to prove beyond any doubt that bile passes out from the gall-bladder through the cystic duct. A crucial experiment which shows that the gall-bladder empties through the cystic duct instead of by absorption of all of its contents has been performed by Copher, who showed that if tetraïodophenolphthalein is injected into a dog after the ligation of the common duct, the shadow of the gall-bladder will remain indefinitely (for three weeks, for example, in one experiment, after which the animal was killed).

Of equal interest with this question has been the one of how the gall-bladder empties itself. It is a curious fact that, in spite of the countless abdominal operations which have been performed, only one surgeon has reported every having seen the gall-bladder contract. Matsuo,[3] a Japanese surgeon, states that once he saw it contract after magnesium sulphate had been instilled into the duodenum during the course of a laparotomy. If the gall-bladder contracts in a manner analogous to that of the urinary bladder or the intestine, then one would suppose that by means of cholecystography it would be easy to demonstrate the contraction waves. As a matter of fact, however, not only my associates and I, but also many others, have patiently watched the visualized gall-bladder through the fluoroscope, but have never seen any suggestion of a contraction wave. Similarly, also, stimuli which cause violent contractions of other hollow viscera in the abdomen elicit no demonstrable response when applied to the gall-bladder. This organ can be pinched with a clamp, incised with a knife or touched with a cautery without the least sign of a contraction wave of any kind. Moreover, as Boyden[4] and Whitaker first showed, an electrical stimulation which will throw the intestine into violent peristaltic contractions will, when applied to the gall-bladder, evoke no response. We have also repeated this experiment many times with similar results. It seems certain from all this, therefore, that if any muscular contractions occur in the gall-bladder they are extremely difficult to demonstrate, and the inference may also be drawn that they are therefore of little importance. The

rhythmic contractions of the gall-bladder described by Bainbridge and Dale[5] and by others are probably nothing more than the effect of pressure exerted by neighboring organs as the result of respiratory movements; at least in the experiments of these authors the possibility of the effect of respiratory movements as a cause of the contractions has not been satisfactorily excluded. Moreover, when the gall-bladder itself is examined it is found that its muscle tissue is relatively scanty. Boyden states: "The muscularis of the intestine is considerably thicker than that of the gall-bladder, the difference being far greater than appears, since the fibers in the intestine are so much more closely packed," but "the connective-tissue layer of the gall-bladder is nearly three times as thick as that of the intestine and, as stained with resorcin fuchsin, is evidently much richer in elastic tissue; this tissue is surely an important factor in the mechanics of the gall-bladder."

Copher and Kodama, in our laboratory, have recently performed crucial experiments, not yet published, which can leave little doubt that whatever active muscular contraction there may be, it can at most play only a minor rôle in the emptying of the gall-bladder. The chief factors in its emptying are purely passive and are explained on simple mechanical principles, as follows: The gall-bladder is a distensible viscus which responds to increased pressures in the common duct by becoming distended. When the ductal pressure is suddenly lowered by a sudden opening of the intestinal end of the duct there is an elastic recoil on the part of the wall of the gall-bladder which results in the ejection of bile from the organ. Also, as the bile is streaming down the common duct past the orifice of the cystic duct, there may be some siphonage action in a manner analogous to that of the filter-pump well known to chemists, although the Heisterian valves may interfere somewhat with the exit of bile in this way. The intermittent sudden opening and sudden closing of the duodenal end of the common duct results, therefore, in a gradual washing out of the gall-bladder. But obviously it can never be entirely empty. Copher has shown that if daily injections of tetraïodophenolphthalein be given to a dog a shadow of the gall-bladder remains throughout the entire period of the experiment. This shows conclusively that the gall-bladder is never empty, at least during the period of the experiment. The intermittent opening and closing of the duodenal end of the common duct is in turn depend-

ent chiefly upon the state of contraction of the wall of the duodenum. Kodama has devised a model to illustrate the filling and emptying of the gall-bladder based on these principles, and a description of it will appear in the June number of the *American Journal of Physiology.*

If, now, these conceptions are correct, then it should be possible to substitute in a living animal an artificial elastic gall-bladder which would act very much like a normal one in emptying and filling. Such was found to be the case.* Copher and Kodama removed the gall-bladder from a normal dog and connected a small rubber bag with the cystic duct, at the same time surrounding the artificial gall-bladder with a celloidin cylinder to prevent pressure on it from other organs. By using cholecystographic examinations with the aid of tetraïodophenolphthalein it was found that this rubber gall-bladder behaved like a normal one, except that of course there was no ability to concentrate the bile by absorption of water, and that a slightly longer time was required for emptying, due probably to the facts that the elasticity of the rubber bag was not quite the same as that of the normal gall-bladder and that also the factor of intra-abdominal pressure was excluded by the use of the celloidin protector.

There is also much reason to doubt the existence of a true sphincter of Oddi at the end of the common duct in the sense usually understood. At least it is not necessary to assume that any such sphincter plays a major rôle in controlling the outflow of bile from the common duct. Copher and Kodama have found that a distinct sphincter cannot always be found apart from the fibers of the muscle coat of the intestine. They considered that instead the normal tonus of the duodenal wall might be an important factor in the resistance to the flow of bile into the duodenum, because they observed discharges of bile from the duodenal papilla coincidently with duodenal peristaltic movements. While they were experimenting further with this point, Burget[6] published his excellent paper containing his conclusions that undue importance has been attached to the sphincter of the common duct, that resistance to pressure in the common duct is offered by the normal tonus of the duodenum and that peristalsis of the duodenum is an

* The details of this work will appear in a forthcoming issue of the Journal of Experimental Medicine.

important factor in emptying the duct by a milking action and by aspiration due to reduced pressure following a peristaltic wave. Carlson[7] had also expressed the opinion that the tonus of the duodenal wall is more important than the so-called sphincter of Oddi.

FIG. 1.—Artificial gall-bladder produced by uniting a small rubber bag to the cystic duct of a dog by means of a glass cannula after the normal gall-bladder had been removed. A celloidin cylinder was then placed around the gall-bladder to protect it from changes of intra-abdominal pressure. Twenty-four hours after an intravenous injection of tetraïodophenolphthalein the gall-bladder is just faintly visible.

It is well known that the common duct passes obliquely through the wall of the duodenum. According to Quain,[8] in the human, it runs obliquely in the wall for a distance of 2 to 3 cm. Practically the same anatomical relationship exists in the dog. Copher and

Kodama,* in work not yet published, have shown that this arrangement constitutes a sphincter-like mechanism which is dependent upon the tonicity of the intestine and makes it possible for intestinal peristalsis to be a factor in regulating the flow of bile from the common duct. They found that the discharge from the common bile duct into the duodenum occurs during the relaxation

FIG. 2.—At the end of forty-eight hours it is plainly visible.

phase of a duodenal peristaltic movement, regardless of whether the duodenal peristalsis is spontaneous or induced by chemical, thermal, mechanical or electrical stimuli. During a period of contraction of the duodenal wall it would seem to be impossible

* This work will appear in a forthcoming issue of the Archives of Internal Medicine.

for the duct to discharge its contents. Several different substances accredited with the ability to make the gall-bladder discharge its contents were tested from this point of view, and the observations warrant the conclusion that any ability of these substances to cause an outpouring of bile into the duodenum rests chiefly upon their power to induce peristalsis in the intestine with the resulting

FIG. 3.—At the end of ninety-six hours it is less plainly visible than before.

milking action of the duct from alternate contraction and relaxation. It is therefore unnecessary to assume the existence of the hypothetical contrary innervation of Meltzer or even of the so-called sphincter of Oddi. It is interesting, also, that magnesium sulphate is much less potent in causing a discharge from the gall bladder than other substances. Boyden and also Sosman, Whitaker and Edson[9] have shown that a fat meal, especially one

containing egg-yolk and cream, has the power of reducing the cholecystographic shadow of the gall-bladder to about one-tenth of its former size within the brief time of one hour and

FIG. 4.—At the end of one hundred and twenty hours the shadow of the gall-bladder has practically disappeared. This experiment shows plainly that it is possible for the gall-bladder to fill and empty itself without the aid of either changes in intra-abdominal pressure or muscular contraction. In this experiment the process of emptying was probably almost entirely due to the washing out of the gall-bladder by a gradual inflow and outflow of bile from the liver. If the factor of changes of intra-abdominal pressure had been present, the gall-bladder probably would have emptied sooner. For further discussion of this experiment, see the text.

forty-five minutes, much more quickly and effectively than magnesium sulphate. Copher and Kodama have found that oleic acid when placed in the duodenum is even more potent in

this respect. Pituitrin causes an outpouring of bile, probably chiefly, if not entirely, because of its production of peristalsis in the duodenum and not because of any direct effect on the gall-bladder itself. Incidentally, also, the relaxation of intestinal tonus was found to be an equally important factor in the emptying of the pancreatic duct.

In this brief summary of some of the newer knowledge of the physiology of the gall-bladder it has been seen that cholecystography has been of definite help in contributing some of these new facts. With your indulgence I propose now to review briefly some of the features of cholecystography as a method of diganosis. It will be recalled that the principles upon which the procedure is based are as follows: An opaque substance is secreted by the liver into the bile, passes into the gall-bladder and becomes concentrated there. A failure to obtain a shadow of the gall-bladder may occur under any of the following conditions: (1) Insufficient amount of the substance reaching the liver; (2) inability of the liver to secrete a sufficient amount; (3) blocking of the cystic duct; (4) failure of the gall-bladder to concentrate the material sufficiently, which may be due either to disease of its wall or to too rapid emptying of the gall-bladder from duodenal peristalsis. The factor of failure of the liver to secrete the dye can be neglected because experience has shown that even in the presence of extensive liver damage excellent cholecystograms can be obtained if the gall-bladder is normal. Pribram, Grunenberg and Strauss[10] have had good visualization of the gall-bladder in cases of clinically diagnosed acute yellow atrophy. More recently Fried and Whitaker[11] have found experimentally that moderately extensive chloroform necrosis of the liver does not prevent good visualization. If the dye has been introduced intravenously, and if care has been taken to exclude peristalsis of the duodenum by withholding food during the period of concentration of bile, a failure to obtain a shadow may therefore be taken as a nearly absolute indication that the gall-bladder is either not concentrating its contents or that the cystic duct is blocked. In our experience it has more often signified the former condition. It is therefore a definite index of function. It has often astonished us to see how slight a degree of gross pathological change is associated with a failure of visualization. We feel, therefore, that cholecystography is a fairly accurate index of the function of

concentration. Likewise, since changes in size of the gall-bladder can be observed, we feel that cholecystography is also an indication of the function of regulation of pressure within the biliary system. It will be recalled that these are the only known functions of the gall-bladder. Cholecystography would, therefore, seem to give us a fairly accurate index of the state of the only functions of the gall-bladder which are known.

Recently criticisms have been raised against the diagnostic value of cholecystography on the ground that sometimes a normal series of shadows is obtained when at subsequent operation pathological changes are found in the gall-bladder. Richter,[12] in a recent article, calls attention to the recurrence of attacks of gall-bladder disease and states that probably in the intervals and in the early stages of disease the cholecystographic examination would show a normal gall-bladder and hence would be misleading. It is possible that this fear may be correct but, so far as I know, there is no data to support it. Our own experience is all opposed to this possibility. It is unfortunate that Richter, like so many others, regards "thick, tarlike bile" in the gall-bladder as pathological. On the contrary, such bile is a normal finding and it is a clear indication of the fact that the concentrating function of the gall-bladder is not impaired. It is our belief, also, that cholecystography, when properly performed, will give evidence of disease before it is recognizable by any method short of microscopic examination of the gall-bladder. This has been our experience repeatedly.

Questions such as these bring up for consideration the more important question of what criteria shall be taken for determining abnormality. Is an organ that is functioning normally to be considered as essentially normal in spite of old evidence of disease? Or are we to conclude that even old pathological lesions—as, for example, adhesions—are to be taken as evidence of active disease demanding treatment? In most of the other parts of the body we are accustomed to a physiological viewpoint. In the case of an old fracture we disregard anatomical abnormality if the function is good; also, we do not consider a finger diseased simply because it may present an old scar as evidence of a previous infection. It is possible, therefore, that a functional test of the gall-bladder, such as is made possible by cholecystography, will really prove to be a more accurate index of the actual condition of the organ

than gross pathological anatomical findings at operation. If this should prove to be the case, cholecystectomy might become less frequent. As judged by our own experience, this would seem to be the case, but I do not think that the method has as yet been sufficiently tested by time to warrant too sweeping conclusions on this point. It would seem safer also to consider calculi in the gall-bladder as potential sources of danger despite the fact that the organ may appear at the time to be functioning normally. It has been our own and others' experience that occasionally a gall-bladder seems to possess nearly normal functions by cholecystographic examination in spite of a clear revelation of calculi. This, however, is an exceptional occurrence. Finally, also, in this connection, I think it should be stated that, at least in the light of our own experience, the utmost caution should be exercised in drawing conclusions of abnormality from results obtained with the oral administration of any of the substances because of uncertainty of the amount absorbed. We have found that the intravenous method of administration of sodium tetraïodophenolphthalein in our last 150 cases has given 97 per cent correct diagnoses as compared with only about 75 per cent with the oral method. All of the removed gall-bladders have been subjected to microscopic examination.

The intravenous method has seemed objectionable to many because of the fear of toxic reactions. The toxicity of the halogenated phenolphthaleins is due largely to the phenolphthalein part of the molecule, rather than to the contained halogen. A substance, therefore, which would give equally good shadows in smaller doses might be expected to be followed by fewer toxic reactions. We have found that the isomeric compound of tetraïodophenolphthalein, namely, phenoltetraïodophthalein, gives equally good shadows in doses only about two-thirds as large as those necessary when the tetraïodophenolphphthalein is used. Moreover, in a series of 103 patients injected with this substance there have been no severe reactions at all.

This substance differs from tetraïodophenolphthalein in that the iodine atoms are on the phthalein part of the molecule instead of on the phenol rings. It possesses also the additional advantage of coloring the serum sufficiently to make it of use as a test of hepatic function in a manner similar to the Rosenthal test.

SUMMARY. 1. The hypothesis of Meltzer, of the existence of a contrary innervation between the gall-bladder and the so-called sphincter of Oddi, has little evidence to support it.

2. The opening and closing of the duodenal end of the common duct depend almost entirely upon tonus of the duodenum, and it is not necessary to assume a separate sphincter to control this mechanism.

3. The spurting of bile through the ampulla of Vater is due largely to the milking action of duodenal peristaltic waves upon the common duct, which passes obliquely through the duodenal wall.

4. The emptying of the gall-bladder is chiefly a passive phenomenon due to factors such as the elastic recoil of a distensible viscus and the gradual washing out of its contents by the ingress of fresh bile from the liver. Increased intra-abdominal pressure is also a factor of some importance.

5. The gall-bladder fails to contract when any of the experimental methods are tried which are known to induce violent contraction of either the urinary bladder or the intestine. Muscular contraction of its wall must therefore play an insignificant rôle in the emptying of the gall-bladder.

6. Those substances which are known to induce emptying of the gall-bladder probably have this property by virtue of their ability to cause duodenal peristalsis. Oleic acid seems to be the most potent in this respect.

7. Cholecystography is a test of the only functions known to be possessed by the gall-bladder. If the organ has been shown to be functioning normally, it almost certainly is not causing symptoms, even if at operation old evidence of disease is found, such as adhesions and scarring. Accuracy in interpretating normality, however, demands great care and experience in performing the test. The intravenous method of administration gives much more accurate results than the oral.

8. Phenoltetraïodophthalein possesses many advantages over tetraïodophenolphthalein, and in our own work we are now using it exclusively.

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DISCUSSION ON THE PAPERS OF DRS. RICHARDSON, C. H. MAYO, BRUCE, McGUIRE, DEAVER AND BURDEN, HITZROT, JUDD, CRILE, DOUGLAS, GRAHAM AND COLE, COPHER AND KODAMA

Dr. Fred B. Lund, Boston, Mass.:

In the dark ages when I studied physiology in the Harvard Medical School we learned a list of the animals which have no gall-bladder, and that was the one thing which I remembered out of the physiology because of the remarkable list. The animals that we were taught at that time had no gall-bladder were the horse, the camel and the ox, the dove, the cuckoo and the whale, likewise the elephant and the ostrich.

Dr. Mayo has extended the list. I do not know whether he said the animals that shed their horns do or do not have a gall-bladder. Anyhow, it appears that other animals have been dissected since that time and very many facts of more or less importance have been ascertained.

There is so much that we are all interested in and know so little about in these papers that it is hard to tell what to speak of. Dr. Deaver alluded, among other things, to the fact that in acute gall-bladder it was a wise thing to wait until the attack had quieted down. Of course, the gall-bladder is not like an appendix; it is not so dangerous to wait, and we generally do wait. At any rate, we have learned not to let a practitioner wait and watch the case at home, because if we watch him in the hospital we have the chance to jump in any day quick when we have to, and we certainly do have to sometimes.

I saw a woman who had a swollen gall-bladder at night, and said I would operate the next morning. The next morning she was much sicker; the tumor had disappeared. When we opened her we found the gall stones distributed about the abdominal cavity, and it was some time before we could find what was left of the gall-bladder way up under the liver. That was the only case where I had perforation occur while waiting to operate. She recovered.

In a number of cases in which waiting has been going on, and the patient gets a little worse, I have found the most wonderful demonstration of Dr. Deaver's theory of the extension along the lymphatics to the pancreas, because while we were waiting, the contiguous parts of the pancreas to the gall-bladder were developing fat necrosis, which we found and took care of. Those cases got well. Lots of cases of pancreatitis with simply fat necrosis without hemorrhage have gotten well in my hands.

I do not know whether I shall ever dare take out a gall-bladder after

looking at Judd's pictures, but the results have been very satisfactory wherever we have removed gall-bladders, even in the presence of a swelling of the pancreas. If pancreatitis does occur I am sure it is by extension in the lymphatics from the liver and the gall-bladder. But suppose the first thing that happens is edema of the pancreas, and the pancreas being shut up behind the stiff peritoneum, the backbone behind it, cannot swell and get soft; it is bound to get harder, just as you would blow up a rubber ball. The harder you blow it up the harder it gets. We feel this hard pancreas, we take out the gall-bladder and relieve the infection and the patient gets well. The pathologist says that thing is not pancreatitis at all. Why cannot it be simply a preliminary stage to a pancreatitis, not a chronic pancreatitis, as we call it, but an acute edema which has taken place during the chronic infective process?

In removing gall-bladders we cure our cases; but there are certain cases where it is a great deal safer, as Dr. Deaver has said, to leave the gall-bladder behind, to simply drain it and, as I grow older, more of the patients in whom I drained the gall-bladder are coming back with recurrences. What good is a gall-bladder left behind from a functional point of view? None at all. It has lost its elasticity entirely. It cannot suck up the thin bile and send out the thick. It cannot move; it cannot do any good at all; gall stones are going to recur in it. But a patient with recurrent gall stones is better than a dead patient.

I have two stout patients now that I have operated upon twice for the removal of gall stones and have not dared to remove the gall-bladder, and if they come again I probably shall not dare to remove the gall-bladder.

You may prescribe exercise for these people. I understood Dr. Graham to say that the movements of the viscera over the gall-bladder do not tend to produce any more drainage. I do not know that they do. We know that the gall stones come in stout people a great deal, and I always believe that indolence is a cause of cholecystitis. However, one acute case of cholecystitis that I saw I thought might be due to indolence, but the patient proved to be one of the hardest-working blacksmiths in the state of Maine. I am inclined to tell these people to move about and exercise. I should think if they would ride horseback about twelve hours a day a gall stone could not possibly form; but it is hard to get fat people to do that.

Dr. Deaver said that gangrene is rare in his experience. I have seen quantities of cases come into the hospital with gangrenous gall-bladders, not entirely gangrenous always, but gangrenous spots on the fundus. Those are the cases where there is a big stone caught in the cystic duct and, as the swelling takes place, the pressure of that stone

shuts off the cystic artery and gangrene ensues. You always get gangrene before you have a perforation, because perforation takes place through a gangrenous spot. At least my experience does not bear out Dr. Deaver's statement that gangrene is rare, because in our town it is very common.

Dr. Carl A. Hamann, Cleveland, Ohio:

I should like to refer briefly to a simple point in the technic after cholecystectomy, the question as to whether or not to drain. We have collected our cases of cholecystectomy since April, 1925, and out of 59 cholecystectomies there were 9 cases in which bile drained subsequent to the operation. The longest drainage was a period of three months and the shortest was for five days. All of them finally closed without any further interference.

You may say that these frequent cases of drainage of bile after cholecystectomy were due to injuries to the common or hepatic ducts. I am as certain as I can be that they were not, because we spend considerable time in isolating the cystic duct. We insist upon the importance of drainage after cholecystectomy.

Another point in regard to the treatment of acute cholecystitis: I believe we should regard acute cholecystitis in the same light as we regard acute appendicitis. We do not know what either of them is going to do. Gangrene is not excessively rare, nor is perforation. Therefore, I think that every case of acute cholecystitis should be operated upon either by drainage or preferably by cholecystectomy, if that is feasible.

Another small point in regard to the anatomy of the under surface of the liver: On a good many occasions we have seen a small fissure, from $\frac{1}{2}$ to $1\frac{1}{2}$ inches in length, on the under surface of the right lobe, about on a level with the middle of the gall-bladder. This fissure is quite insignificant; it is of no importance morphologically that I know of, but it has not been described so far as I know.

Dr. J. Shelton Horsley, Richmond, Va.:

Many excellent things have developed in this symposium, but because of personal experience I was particularly interested in the question that was brought up by Dr. Bruce, on the coincidence of pathology of the gall-bladder with pathology in the duodenum. If lesions of both of these organs are initiated by hematogenous infection, as we are led to believe, along with hepatitis, as Graham has demonstrated, it may well be that what appears as single, as, for instance, duodenal ulcer, may be merely a survival of multiple original lesions, the remainder having recovered.

In the early stages of the development of the pyloroplasty I have been doing for some years, among other errors committed, I sutured the pyloric mucosa. Dragstedt[1] has shown experimentally that an excellent way to produce a peptic ulcer in the pyloric region of a dog is to apply multiple sutures at any one point. I found that a considerable proportion of the cases of pyloroplasty in which I sutured the pyloric mucosa had a recurrent ulcer, apparently due to this reason. Since abandoning suturing of the mucosa in the technic this trouble seems to have been so far avoided.

I would also often merely separate the adhesions of the gall-bladder to the duodenum, but I found that in these cases in which the adhesions were separated and the gall-bladder left there was often a recurrence of the symptoms; on reoperation if there was no recurrent ulcer the symptoms usually cleared up if the adherent gall-bladder was removed.

Adhesions, of course, like malaria, liver complaint and worms, cover a multitude of sins, but they certainly do mean something in certain regions. In regions in which there is normally a rather limited motion, adhesions will probably produce symptoms. For instance, if the heart is adherent to the chest wall the whole mechanism of emptying the heart has to adjust itself toward this fixed point. That causes, as we know, pronounced symptoms. In the duodenum, where the motion is active, and the range of motion is rather limited, a fixation of adhesions to the gall-bladder may produce symptoms, whereas in a more mobile transverse colon or in the loops of the jejunum, adhesions, unless they cause active obstruction, will probably give no symptoms. The relief of these adhesions frequently relieves the symptoms. We have all seen cases in which there were fixed adhesions to the duodenum that would produce almost precisely the same symptoms, even so far as the roentgenologic examination is concerned, as would a duodenal ulcer.

In pyloroplasty the normal current of food is reëstablished, and this throws work upon the duodenum that is temporarily somewhat incapacitated because of the trauma of the operation. In a gastroenterostomy, however, particularly if the pylorus is closed, there is a large artificial opening and the duodenum is put at rest; consequently there is no occasion for marked peristalsis, either in the stomach or in the duodenum. For this reason an adherent gall-bladder may be left in a gastroenterostomy and will not give symptoms, particularly if there is a stenosis or a potential stenosis, while adhesions in a pyloroplasty, particularly to the gall-bladder, will usually cause symptoms.

[1] Gastric Ulcer Studies, Arch. Surg., 1924, viii, 791.

Dr. Edward W. Archibald, Montreal, Canada:

In a symposium that has covered so wide a field one can only, in discussion, take up points that have particular personal interest. In this excellent series of papers, that of Dr. Graham happened to interest me most. We must all congratulate him on his ingenious theory and no less ingenious experiment. Dr. Graham, if I understood him rightly, was of the opinion that the gall-bladder, though it had a few muscle fibers in its walls, possessed really no muscular action, or, if so, a negligible one. He also suggested that our old friend, the sphincter of Oddi, did not in reality exist, or that if it existed, quite frequently it did not exist; you understand the paradox.

Of course, these two things have been pet protégés of my own, and I must rise in their defense. It may be perfectly true, as Dr. Graham says, that nobody, with one exception, has ever seen the gall-bladder contract. His one exception I did not catch. I admit that I, too, have tried often in experimental work to get the gall-bladder to contract, and have failed, save on one occasion, about which I shall speak in a moment. Nevertheless I cannot yet admit the truth of such a negative argument. Is the sphincter of Oddi to become a sort of anatomical "Mrs. 'Arris?" Perish the thought! Muscle is there. It must contract. Let us look a little deeper into the question. One must remember that patients and animals are practically always under deep ether anesthesia, which abolishes function. If you take a contractile organ like the stomach, how often do you see it contract in a patient under ether? It rarely does. You can sometimes provoke a limited contraction by stimulation, but one rarely sees the ordinary peristaltic contractions except when operating under gas-oxygen anesthesia. But this is an argument drawn from analogy.

There exists in the body a number of sphincters—the esophageal, the pyloric, the anal, the vesical, the ureteral, the ileocolic and the sphincter here in question, that of the common duct, or sphincter of Oddi—of the presence of which in animals I have no doubt, having seen it frequently in action and verified its presence by histological examinations. In the stomach, of which our knowledge is most precise, we find behind the pyloric sphincter a section, comparatively small, whose function lies in peristaltic muscular action; behind this the body of which the muscular action is not peristalsis, but tonic contraction, a contraction of all the muscle fibers equally so as to produce a gradual concentric diminution of the organ's capacity, but only in moderate degree. Here, then, we have a sort of system—a tonically contracting bag, forcing contents quietly and slowly into an actively working peristaltic portion, which leads up to a sphincter outlet. Somewhat the same plan is seen in the other hollow organs mentioned, leading to exits

which are guarded by sphincters. This general idea I have applied theoretically to the anatomical system of gall-bladder and bile ducts. Behind the sphincter of Oddi there lies the peristaltic portion of the common duct and probably the cystic duct and neck of the gall-bladder, and behind this lies the tonic muscular bag of the gall-bladder itself. Upon this theory, one should not expect to see the gall-bladder actively contract. If it were filled to a certain degree of distention, a degree suitable to the excitation of a motor-nervous response, and if this action were not prevented by an anesthetic, and if the hormone stimulus happened to come along at the same moment, then one might observe a slow, tonic contraction of the gall-bladder wall, the only evidence of which would lie in a quiet diminution in size of the distended organ, easy to overlook. It is indeed no wonder that Dr. Graham and others are led to deny any real evacuating action on the part of the gall-bladder and to seek elsewhere for reasons to explain its undeniable emptying. But are there not many here who, in the course of operations, have been surprised to find that the full gall-bladder which they had observed upon opening the abdomen was at the closing nearly empty? I, at least, have been struck by such an observation not infrequently. But, coming to something more definite, I find that I am able to go further and to say that I have actually observed, in a cat under experiment, the gall-bladder to contract in this slow, tonic way. This question of gall-bladder contraction, I may say in parenthesis, has engaged my attention only casually during the course of other experiments. In cats, and in a few dogs, I have tried faradism, mechanical irritation and the application of hot water, and have always failed to get a response in the way of a visible contraction of the gall-bladder wall. Finally, however, I tried the application of a weak solution of barium chloride (I speak from memory and have not the exact figures). It is known that this substance will provoke gastric contractions when other kinds of stimulus fail. In one out of two or three attempts I succeeded in calling forth the response just described. The gall-bladder contracted within a few moments to about half its previous size. The question must be further investigated in animals operated on under local anesthesia.

As to the sphincter of Oddi, it certainly exists, and is active in dogs and cats at any rate. You cannot open the duodenum and run a solution into it through the gall-bladder under normal and raised pressures without seeing the sphincter alternately give way and contract. You cannot under the same circumstances apply a weak solution of hydrochloric acid to the duodenal mucosa, or pinch that duodenal mucosa, without noticing a contraction of the sphincter in the duodenum which suddenly blocks the flow of the solution. You can

actually see the pouting of the papilla, and then the contraction of that pouting tip upon the sphincteric closure, on the application of these things. One simply must conclude that there exists at that point a sphincter.

The question remains, what measure there may be of truth in Dr. Graham's explanation of normal gall-bladder emptying. I cannot think that the experiments, ingenious to a degree as they are, prove his point or exclude muscular action of the gall-bladder. His theory is of the sort that one cannot disprove. And one may imagine that Dr. Graham himself is not inclined to insist strongly upon the "milking" analogy. The old cow does not let down her milk to the unpractised hand. I possess no exact knowledge of the physiology or the mechanics of the process of milking, but I do not in any case believe it acts by suction or siphonage. There is something organic or vital in the cow, and I suspect also in the bile system, which cannot be relied upon to allow milk to come down, or bile to come down, except at the call of a physiological stimulus. Some reflex has to be started. In milking there is always the preliminary stroking, then the first small stream of milk and, finally, with a good compression of the terminal ducts well up upon the base of the udder, the easy and full flow, suggesting an actual expulsion of milk from the udder. In the human the flow of bile is apparently excited by the entrance of food into the duodenum, just as is the pancreatic juice. Is it not still reasonable to believe that the muscle of the gall-bladder is reflexly stimulated to expel the bile? This seems to me more physiological than to postulate a purely mechanical suction action.

Dr. Philemon E. Truesdale, Fall River, Mass.:

Dr. Graham's recital and demonstration of the forces which play a part in emptying the gall-bladder were very fascinating. I presume he had considered the useful function of intraäbdominal pressure in this connection, although no direct reference was made of it. Surely it seems that intraäbdominal pressure plays an important part in emptying the gall-bladder.

As Dr. Mayo said, the gall-blader is full in the morning after a long period of fasting, and empties during the process of digestion. Thus with the intake of food and the resulting increase in the intraäbdominal pressure the normal gall-bladder empties. One thinks of this force in connection with emptying the gall-bladder, particularly because the positive pressure within the abdominal cavity applies a force upon everything within its boundaries. While this pressure within the abdomen is constant, it probably finds its lowest degree during periods of fasting. Faulty digestion with considerable fermentation no doubt interferes with the normal filling and evacuation of the gall-bladder.

I would be interested to hear Dr. Graham touch upon this phase of the problem in his answers to questions already proposed.

Dr. Ellsworth Eliot, Jr., New York City:

I wish to confine my remarks to the treatment of stricture of the common duct. I think that this subject can be most conveniently divided into three different types: (1) Efforts by some form of plastic operation to replace a missing segment of the duct; (2) an effort to reëstablish the continuity of the duct, perhaps assisted by some partial or incomplete plastic operation; (3) an effort to establish anastomosis between the stump of the duct and the intestines.

In this last classification I should like to call attention to the fact, and emphasize it, that in such cases the anastomosis should be made preferably between the proximal stump of the duct and mobile part of the intestine, as near as possible to the normal orifice of the duct in the duodenum. Do not go down into the intestine; preferably go up into the stump. An anastomosis between the end of a hepatic stump and the stomach is far preferable and very much less likely to develop a subsequent infectious cholangeitis than an anastomosis between the stump and the upper jejunum. The farther the anastomosis is below the orifice of the normal common duct, the greater the danger of ascending infection.

My remarks chiefly are directed to the effort, in old cases, to be sure to establish the continuity of the duct in strictures resulting from damage to the duct in a previous cholecystectomy, as well as in those cases of simple stricture with the gall-bladder still intact, a much rarer though still a possible condition. Strictures situated above the level of the duodenum are obviously more favorable to repair than those in the lower part of the common duct or those in the hepatic duct before its exit at the transverse fissure. In this group of supraduodenal strictures it is usually possible after excision of the cicatricial tissue to approximate by suture the divided ends of the duct, if the interval to be bridged does not exceed $\frac{1}{2}$ or exceptionally $\frac{3}{4}$ inch.

I refer in this connection to the oldest case within my own personal observation of a stricture relieved in this way, where the distance between the ends of the duct amounted to $\frac{3}{4}$ inch, and where it was possible only to approximate the posterior edges of the divided ducts, the gap anteriorly being closed in by a flap of peritoneum from the adjacent gastrohepatic omentum.

After the repair was made a soft, flexible T tube was inserted, extending above and below, and left *in situ* for a month. For only twenty-four hours after its removal was there any leakage. [1]

[1] For full history of this case see Trans. Am. Surg. Assn., vol. xxxv, pp. 247, 248.

This patient, eleven years after the operation, is alive and well, and with the exception of several slight attacks of pain and jaundice during the first year after operation has had no further trouble.

I want to refer briefly, if the time allows, to the most recent personal experience of a stricture following a cholecystectomy, in the hands of another surgeon, that was so close to the transverse fissure of the liver that it was impossible to mobilize the stump of the hepatic duct. In this case after the removal of the cicatricial tissue the gap was closed by a peritoneal flap brought up from the gastrohepatic omentum, after it was seen that a suitable flap could not be procured from the adjacent visceral peritoneum of the liver. A similar tube to that described in the previous case was then inserted, and after its removal at the end of a month a slight biliary discharge for forty-eight hours was observed with prompt closure of the fistula. While this case cannot be considered an end-result, the primary result has at least proved most satisfactory.

Dr. Miles Fuller Porter, Fort Wayne, Ind.:

I have some doubts as to whether I correctly interpret what I have heard. If my interpretation is correct, I have heard, it seems to me, one of the strongest arguments I ever heard in my life against the routine removal of the gall-bladder. If the disturbance of Byron Thompson's abdominal brain induces stricture of the duct, if handling of the common duct and the hepatic ducts is necessary to an ectomy, which handling is not necessary if a cholecystotomy is done, then it seems to me that in this argument alone we have a sufficient one for not removing the gall-bladder without sufficient cause.

A further illustration in Dr. Judd's case seems to me to be the actual demonstration of the fact that apparently the principal trouble still remained in the cholangeitis inside the liver after the gall-bladder was removed.

I should like to have Dr. Judd and these other men tell us how many of these cases of subsequent stricture of the common duct follow the cholecystotomy.

I should like to say one word, too, about the use of the T tube. I may be entirely wrong, but with the exception of the use of the T tube under circumstances advocated by McArthur, in my own humble opinion the T tube has no business in the surgery of the common duct when we operate upon it for the purpose of drainage. Instead of the T tube introduced inside of the duct, if we use a tube with the end cut out, sort of a fishtail, I think W. J. calls it, and put it over the opening, there is no irritation to the internal lining of the duct.

My experience, as compared with the experience of these men who have talked, has not, of course, been great, but I never have seen a stricture of a duct follow this sort of drainage; neither have I seen a stricture of the tube follow a cholecystotomy wherein there was not already existent a clinical patent advanced cholangeitis at the time of the operation.

I should like to mention, too, the question of whether we should operate or whether we should wait regarding acute cholecystitis. If a patient dies as a result of infection of the gall-bladder, why does he die? He dies because of extension of the trouble to the liver, or perhaps on to the kidney, or perhaps on to the pancreas, or perhaps as a result of perforation, and it takes time for all of these things to happen. Why not save the time and thereby the patient by opening and draining the gall-bladder?

Dr. Alexis V. Moschcowitz, New York City:

I greatly regret that I cannot quite agree with the advocates of a complete closure of the wound after cholecystectomy. I have so frequently seen leakage of bile for a few days after this operation that I confess to a certain timidity in closing the wound completely. Nor do I see any urgent indication for the same. Even with a very liberal drainage, the kind I usually employ, the wound is usually entirely healed in a little over two weeks, which is certainly short enough to satisfy even the most fastidious patient. If, in addition, a transverse incision is used, the danger of hernia formation is negligible. On the other hand, I have discarded for a number of years all forms of intraductal drainage in my cases of choledochotomy. The common duct is either open at the termination of the operation, and if so it requires no drainage; or it is closed, and if so, it is the duty of the surgeon to see to it that it is open.

Some years ago I had a very distressing experience after a very simple cholecystectomy. Shortly after the operation the patient developed a complete biliary fistula. After four months I persuaded her to let me attempt its closure. At this operation I found a short stump of hepatic duct, but nothing distally. After a search for the common duct, which lasted four hours, I abandoned it (this only bears out what most of the speakers said about the difficulty of closing these cases) and satisfied myself with a hepaticoduodenostomy. The patient again developed a complete external biliary fistula which lasted for over three years. At this time I persuaded the patient to permit me to try to close it again. While the patient waited to be admitted to the hospital (about one week after her last visit to me) the fistula suddenly ceased draining, the external opening closed and has remained so ever since that time, which is now a period of about two years.

Dr. Mayo spoke of the gall-bladder of 1926. Unless I am greatly mistaken, we have discovered it also at Mount Sinai Hospital, only we have given it a different name. Most of the time there is nothing wrong pathologically with these gall-bladders. Their main characteristic is, that if the surgeon removes them, in spite of this negative indication, the patient is not only not benefited, but distinctly harmed by the procedure.

Dr. Richardson (in closing):

I have nothing to add, Mr. President, except to thank the gentlemen for their discussion.

Dr. McGuire (in closing):

There has been a good deal of discussion this afternoon regarding the injury to the common duct. It seems that the answer is that, instead of trying to treat the injury to the common duct, we ought to say something about preventing it. It is a perfectly simple thing to prevent in a great majority of instances. The trouble is that a great many men do cholecystectomies who do not do them often, and they follow the lead of men, like Judd, who tell them they have to do them a certain way. Judd tells them they have to do it from below upward. That is all right for Judd; it is all right for a good many others, if they dissect it out as they should and clamp the cystic duct where they should.

I think the advice ought to go to people who are not as well versed as you are that they do not have to be so careful about leaving a part of the cystic duct. Leave a little gall-bladder if you have to; it is not going to do any harm if you leave a small amount of gall-bladder; then you know you are not going to injure the common duct.

I am perfectly convinced that there is quite a series of cases where it is very difficult to see that cystic duct, and it is very much easier to do cholecystectomies from above downward in those cases. I do not say it should be done often, but it should be done particularly in those cases where it is going to be difficult to get a view of the cystic duct, so as to be sure of not injuring it. I think the cure is prevention rather than trying to reconstruct it after injury.

Speaking of livers, a short while ago I had a gentleman in whom I found a large common-duct stone, a stone as big as a small egg. The stone was removed under local. The man also had stones in his gall-bladder. He went through the operation without any difficulty, but following the removal of the stone there was a gush of rather turbid fluid for a period of a minute or two, and after that until he died, three days afterward, there was no bile escaped. I think that as far as the

liver is concerned it has some function that we do not know about. I think that man died from liver insufficiency. When they are jaundiced over a certain period of time, I think there comes a time when the liver does not recover and those people go on and die.

Dr. Crile, a number of years ago, told about some experiments, putting air-pressure on the medulla. Even though the centers were dead, the pulse went on. As far as the liver is concerned, I believe there comes a time when it is dead, and there is no possibility of its recovering, even though the patient lives two or three days afterward.

Nobody has said anything about acute hepatitis. I believe it is a real disease. I think it is something different from biliary cirrhosis. Dr. Bevan reported a case this afternoon where he showed there was no obstruction to the common duct after he had operated a couple of times. I think that was a case of infection of the liver, hepatitis. That liver was damaged beyond repair. We have had several cases where the man did not have any evidence of disease of his gall-bladder; he was operated on, and he went on and died, and nothing was shown except infection of the liver itself without any disease of the rest of his biliary system.

Dr. Deaver (in closing):

These papers have been so well discussed that there is practically nothing to add.

As to the question of acute perforation, I can repeat that I see comparatively few cases of acute perforation in comparison to the great number of cases of gall-bladder disease.

I do, however, see a number of cases of chronic perforation where Nature has protected the peritoneum against peritoneal disaster. Where we are reasonably sure of acute perforation there is no question about the advisability of immediate operation; also in partial gangrene of the gall-bladder. This is the type of case where surgery had better not be any more radical than it can possibly be. In a number of instances I have removed the fundus of the gall-bladder, left the lower portion which was not involved in the gangrenous process, put in a drainage tube and the patient has gotten well.

In the infected gall-bladder with surrounding infection cholecystectomy may open avenues favoring the spread of the infection, defeating Nature's efforts at coffer-damming.

Another point which should influence us is the degree of peritonitis: I make it a rule in gall-bladder disease, as I do in appendicitis, where there is a diffuse peritonitis to administer anatomic and physiologic rest.

I cannot agree with my friend Porter as much as I should like to

on the question of the T tube. I have used this form of drainage in many cases and I have not seen any ill-effects. I have reopened cases that have worn T tubes for four, five or six months and longer, and found the common duct in no way damaged. I have had one patient wear a T tube for four years.

A very distinguished citizen of Philadelphia, now dead, wore a catheter for an enlarged prostate for eleven years, during which time he never voided a drop of urine voluntarily. This gave me encouragement to insist upon the retention of the T tube in the common duct for a long period.

Dr. Hitzrot (in closing):

I have nothing to add in closing, Mr. President.

Dr. Judd (in closing):

Dr. Lund spoke about being in great fear of ever taking out another gall-bladder after seeing the picture that I showed of the change that occurred in the ducts after removal of the gall-bladder. Of course, the answer to that is that we do not remove normal gall-bladders. It does not make any difference how the gall-bladder is destroyed, inflammation can destroy it just as well, and produce just exactly the same changes in the ducts. I do not think, from this evidence, we need have any more fear of removing the gall-bladder than we had before.

Dr. Porter asked if we had ever seen a stricture of the common duct following a cholecystotomy. We have seen several of them. Of course, if our theory of this obliterative cholangeitis is right, they occur following cholecystotomy and before any operative procedure at all.

Dr. Crile (in closing):

There is little for me to say except that I should like to caution any one who might be inclined to try diathermy. I forgot to mention as a precaution that it is very necessary, if your patient is under anesthesia, not to use rigid plates, as they will burn the patient. Use a mesh.

I should like to say one word about Dr. McGuire's point regarding the possibility that the liver cells may be damaged beyond repair at the time of the operation. I suppose you all will recall that the liver undergoes autolysis within just a few minutes after its removal from the body. The liver cells are extremely sensitive and break down as quickly as, if not more quickly than, any other cells in the body.

I should like to ask the acting chairman whether he and his associate in their large series of cases of cholecystectomy without drainage have noted fewer clinical disturbances afterward as compared with previous series in which drainage was employed? For ourselves we are noting

that since using Morrison's drain and keeping the drainage tubes away from the gall-bladder region we have less postoperative disturbance.

Dr. Judd:

We have had a good deal of experience in closing without abdominal drainage following cholecystectomy. In answer to Dr. Crile's question as to the immedate results, I think I can say that the patients have been very much more comfortable where we have closed without drains. The ultimate results has also been very satisfactory.

Of course the objection to closing without a drain is the necessity of being sure that there will not be any drainage of bile afterward. We figured that we had about 1 in 20 clean cases that drained bile sometime during their convalescence following cholecystectomy, and that kept us from adopting the plan of closing without a drain. We use a great deal of care in selecting the cases, and if there is much edema or infection in the tissues in the field we use drainage. We have, as I say, closed several hundred cases without an abdominal drain and with more satisfactory results so far as the convalescence is concerned than in those that were drained. Personally I cannot see any objection to closing without a drain in a perfectly clean case, as there is no difference between that and closing similarly following a gastroenterostomy.

In the last year I have seen reports in the literature of cases that had these anomalous ducts coming down from the liver directly to the gall-bladder. In these cases one would ligate the ducts in the course of the cholecystectomy. Personally I have not had any such experience that I know of, nor a cholecystectomy case closed without a drain that opened up and drained bile. As stated, we used drains routinely following cholecystectomy, and found that about 1 case in 20 drained bile at some time during convalescence. In view of the fact that we have not had any such experience with cases closed without drainage, it is reasonable to assume that the drain produces sufficient irritation in the surrounding tissues to cause opening of the duct and drainage of bile. This seems the only basis on which we are able to explain our present results.

Dr. Graham (in closing):

There are several questions that have been asked that I should like to speak of for a moment, and then I should like to show two or three slides.

First of all, concerning the question raised by Dr. Truesdale, the relationship of intraäbdominal pressure to the emptying of the gall-bladder. This is an important consideration, of course. It is one of the things which I thought I mentioned this morning in connection with the

influence of respiratory movements, the pressure on adjacent viscera caused by movements of the diaphragm. The point I wished to emphasize this morning was that it is perfectly possible for the gall-bladder to empty, for an artificial gall-bladder to empty, without any reference whatever to abdominal pressure, respiratory pressure or any contraction caused by muscular contraction. The reason we put these celluloid protectors around the artificial gall-bladders was to rule out the factors of abdominal pressure, pressure from respiratory movements and so on. The question also has come up (not in this discussion, but privately) about the waves which have been described, the periodic contractions which one can see if one puts a monometer into the gall-bladder. These periodic contractions we think are largely respiratory movements transferred from the diaphragm to the wall of the gall-bladder by pressure of adjacent viscera. If one paralyzes the diaphragm on that side these movements practically entirely disappear.

Another point concerns the question raised by Dr. Archibald, namely, what does this muscle in the gall-bladder do anyway? That was not the way he put it, but I think that is what he meant to bring out. We believe that this muscle probably aids in tonus of the gall-bladder. Every organ has to have tonus. There is something necessary to keep up tonus. Functions ordinarily cannot be carried out in a hollow viscus unless there is tonus. We believe that this muscle helps to keep up the tonus of the organ, helping also to prevent overdistention, and so on. With reference to the question brought up by Dr. Archibald, there is another point which really is not concerned, perhaps, in the matter that I gave this morning, but which I feel ought to have some statement made about it. Some years ago, about five or six or thereabouts, at the Toronto meeting, we presented a paper suggesting an hypothesis of the lymphatic origin of cholecystitis and pancreatitis, the latter part supporting Deaver's contention that the lymphatics from the gall-bladder pass into the pancreas. You will recall that at the meeting two or three years ago Dr. Archibald questioned these findings. Very recently Kodama, a Japanese, with very excellent technic, at our suggestion, has gone over all this work again, and he finds evidence, at least so far as injection experiments are concerned, that the lymphatics from the gall-bladder do not enter the pancreas, merely that they pass along anterior to it. I feel it is only fair to Dr. Archibald to make this statement here at this meeting. I will not, however, draw the fire of Dr. Deaver onto my head by saying that pancreatitis is not due to lymphangitis, but I simply make that statement so far as injection experiments are concerned. Kodama's work on the extrahepatic lymphatics has shown also very clearly that, so far as injections are concerned, there is a direct lymphatic connection between the first

portion of the duodenum and the gall-bladder and *vice versa*. This, perhaps, might tend to explain an association between duodenal ulcer and cholecystitis.

I want to say a word or two about the clinical use of cholecystography. Various criticisms have been made against cholecystography, and I think quite justifiably, on the assumption that cholecystography would reveal only rather extensive lesions of the gall-bladder and could not reveal early and moderately severe cases of cholecystitis. This assumption is based upon the false premise that cholecystography is an indication of the exact pathology present. On the contrary, what I was trying to lead up to this morning indicates that cholecystography is a functional test of the gall-bladder preëminently, rather than an indication of exact pathology. If we hark back to the days of Galen and consider loss of function as one of the cardinal signs of inflammation, we think that a means of determining function of an organ is pretty likely to be a good indication of inflammation in that organ. Here is a drawing made at operation on a patient who had only a slightly good gall-bladder history, only a suggestive gall-bladder history. On cholecystographic examination, however, by the intravenous method, which is the method that we prefer, no shadow of her gall-bladder was obtained at all. We, therefore, diagnosed her case without much hesitation as a case of cholecystitis presumably. At operation we found a gall-bladder which had slate-blue walls, very thin; nothing whatever could be felt in the gall-bladder; there was no visible thickening. The only pathologic indication of disease was this little adhesion beneath. On the basis of loss of function as shown by cholecystography, however, we removed that gall-bladder; the patient promptly got well, and she has been well for one year. Incidentally, also, I might say that microscopic examination of the gall-bladder showed a good deal of leukocytic infiltration of its walls.

Here is another one somewhat similar. It failed to show any shadow whatever. The only two functions of the gall-bladder that are known are shown by cholecystography, because cholecystography, by showing changes in the size of the gall-bladder, will illustrate the regulation of pressure, which is one function, and by changes in density will illustrate the power of concentration, which is the only other function known that the gall-bladder possesses. Here we found a gall-bladder almost identical to the other except for these few adhesions. This gall-bladder was removed a year ago. The patient also has been entirely free from symptoms every since, indicating again, I think, that cholecystography will detect relatively minor changes in the gall-bladder if the function is disturbed.

We are now using not tetraiodophenolphthalein, but its isomer,

phenoltetraiodophthalein, in which the iodine atoms are united to a different part of the molecule. We find this has many advantages over the tetraiodophenolphthalein. It can be given in smaller doses, and we have had no appreciable reactions in over 100 patients to whom we have given it intravenously. It also gives good cholecystograms a little more quickly than either of the other substances which we have used. To those interested in functional tests of the liver, based on the elimination of dyes through the liver, it works perfectly well, just as well as phenoltetrachlorphthalein or any of the other substances which you can use.

I merely want to show two slides showing stones by cholecystography because they have interested me very much indeed, particularly in the light of much of the discussion which has gone on here today. Here is a gall-bladder which by means of cholecystography shows a good function, yet it contains stones. Is this gall-bladder diseased? I do not know. We took this gall-bladder out, however, because we believe that at least gall stones are potential dangers and should be taken out. Here is another gall-bladder which shows a good concentrating function despite the fact that it has a stone. I show these slides particularly because I think if we have an accurate evidence of function of an organ, perhaps we can tell more about it than we can tell merely by palpation or even by naked-eye examination at operation.

A STUDY OF THE MORTALITY IN APPENDICITIS

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Mr. Bland-Sutton, in his book on *Gynecology*, has this introduction: "When surgical authors are able to restrain their vanity and refrain from publishing notes of successful cases in textbooks, the established facts of the art can be presented in a very convenient compass." My hope is that the present list of cases will not be unduly long nor unduly uninteresting. I am also conscious of the fact that there is much truth in the aphorism that statistics can be made to tell anything, even the truth. They are of value only in-so-far as their meaning is correctly interpreted.

The present study is based on a consecutive unselected series of 2959 cases with 16 deaths, or a mortality over all of 0.54. It will be worth our while if we can find the real meaning of these figures.

I do not think that I have attended a surgical meeting in ten years in which the question of appendicitis has been discussed, nor have I attended a surgical meeting in ten years in which the question of gastric and duodenal ulcer has not been discussed.

The following are five reasons, I believe, that justify my presenting to the American Surgical Association a paper concerned with the mortality in appendicitis:

1. Appendicitis is still by far the most important acute abdominal disease that surgeons are called upon to treat.

2. During the year 1925, in the United States and Canada, there probably were about 500,000 cases of appendicitis. This figure, of course, is an approximation; it is not literally true; it is not necessary that it should be literally true.

3. During the same year and in the same area there were approximately 25,000 deaths from appendicitis.

4. The death-rate from appendicitis equals the combined death-rate from ectopic pregnancy, pyosalpinx, gall-stones, pancreas,

spleen and the thyroid gland. It nearly equals the mortality from gastric and duodenal ulcer, intestinal obstruction and gall-stones.

5. From a study of the figures available at the Government Bureau of Vital Statistics, it is clearly obvious that the death-rate has increased from 11 per 100,000 of the population in 1920 to 14.4 per 100,000 of the population in 1925. This indicates a gradual rise of over 30 per cent.

The study that we are making does not represent the composite work of some group or clinic, but has solely to do with my individual work throughout the past twenty-five years.

So much then for the background on which this study is based. We will ask you to consider the study as found in Group I.

	No. cases.	No. deaths.	Mortality.
Chronic appendicitis	1241	0	0.0
Acute appendicitis	688	1	0.15
Gangrenous, ruptured localized abscess	570	4	0.7
Acute diffuse peritonitis	85	7	8.2
	2584	12	
Cases in extremis abscess drained, appendix not removed	9	3	33.33
	2593	15	0.58

(*a*) We find that there are 1241 cases of chronic appendicitis operated on with no deaths. Someone has said of this group of cases that there were only two kinds of appendicitis; acute appendicitis and appendicitis for revenue only. In my opinion, this statement contains more of wit than of wisdom. If there is such a diseased entity as an acute inflammatory process in the appendix, then there must be such a condition in which the acute process has become chronic; else how would you classify that large group of cases which have had a definite inflammatory outbreak, the acute attack subsiding, but the patient never completely recovering until the so-called interval operation is done, demonstrating an appendix that shows all of the ear-marks of an inflammatory process that was once acute but is now more or less chronic? It seems to me just as logical to find a chronic inflammatory process in the appendix as it is to find it in the gall-bladder, pancreas or elsewhere. Call it by whatever name you will, all of you know

precisely what I am talking about. Of course, we do not expect mortality in this group of cases. When mortality does occur, it is the mortality of some unforseen and unforseeable calamity.

In a series of cases of this number some of them will be found to tax one's surgical skill greatly. In the average case the operation is extremely simple: in large, stout people, with the appendix deeply placed and inaccessible, the story may be quite different.

(*b*) Consider the acute cases. By the word acute we mean exactly what the term implies. Only the cases are here included that showed definite, unmistakable evidence of an acute inflammatory disease.

In this group is included every acute case up to the point of rupture. When the appendix was ruptured, the case was included in the group of localized appendiceal abscess or in the group of acute diffuse peritonitis. In most cases of the acute group there were gangrenous patches, varying in degree, present in the appendix. In this group there were 688 cases with 1 death, a mortality of 0.15 per cent.

(*c*) There were 570 cases of gangrenous ruptured appendices in which the inflammatory process had become clearly localized. In this group there were 4 deaths; a mortality of 0.7 per cent. You will readily understand that in a group of localized appendix abscess cases of this number one would very nearly run the gamut of intraperitoneal suppuration. The abscess cavity would vary in size from that of a hickory nut to one that would extend from the cul-de-sac of Douglas to the kidney fossa and above, reaching at times into the left lower quadrant of the abdomen. I was quite surprised to find that in this group of cases there were only 4 deaths.

(*d*) There were 85 cases of acute diffuse peritonitis seen for the first time in this stage of the disease in which the operation was done immediately that the diagnosis was made. There were 7 deaths in the 85 cases, or a mortality of 8.2 per cent. Later on we will have more to say about the management of the cases in this group, for on their management hinges in large part the question of the mortality rate.

(*e*) This is a small group of 9 cases, in which the patients were in extremis from long-continued suppuration. They have been separated for the reason that they were the only cases in the suppurative group in which the appendix was not removed and

drainage was instituted under local anesthesia. One of the points of interest in this small group is whether or not there was any relationship between the death-rate of 3 deaths in the 9 cases and the fact that the appendix was not removed.

It is significant that these 9 cases are the only ones in the entire series in which the appendix was not removed.

We have, then, a total from the first grouping of 2593 cases with 15 deaths, or a mortality of a bit over half of 1 per cent.

Next we will consider Group II.

	No. cases.	No. deaths.	Mortality.
Acute appendicitis	688	1	
Gangrenous, ruptured, localized abscess	570	4	
Acute diffuse peritonitis	85	7	
	1343	12	0.8
Cases in extremis; abscess drained, appendix not removed	9	3	
	1352	15	1.1

The principal point in this group is that the so-called chronic or interval cases have been omitted from consideration.

We find that there were 688 acute cases with 1 death; 570 gangrenous, ruptured appendices, with localized abscess, with 4 deaths; 85 cases of diffuse peritonitis with 7 deaths; or a total of 1343 cases, exclusive of the chronic group, with 12 deaths, or a mortality of 0.8 of 1 per cent. To the 1343 cases we add the small group of 9 cases with 3 deaths, making a total for this entire group of 1352 cases with 15 deaths, or a mortality of 1.1 per cent.

We will now consider Group III in a study of the mortality rate.

	No. cases.	No. deaths.	Mortality.
Gangrenous, ruptured, localized abscess	570	4	
Acute diffuse peritonitis	85	7	
	655	11	1.7
Cases in extremis, abscess drained, appendix not removed	9	3	
	664	14	2.1

You will see at once that both the chronic and the acute cases have been eliminated from consideration. This leaves for our study only the cases complicated by the gross appearance of pus.

We have 570 gangrenous, ruptured appendices with abscess, with 4 deaths; 85 acute diffuse peritonitis cases, with 7 deaths: making a total of 655 cases, with 11 deaths, or a mortality of 1.7 per cent. Adding the small group of 9 cases that were in extremis, makes a total of 664 cases with 14 deaths, or a mortality of 2.1 per cent.

A study of Group IV is as follows.

	No. cases.	No. deaths.	Mortality.
Gangrenous, ruptured, localized abscess	570	4	0.7
Acute diffuse peritonitis	85	7	8.2
Acute diffuse peritonitis, deferred operation	123	2	1.6

There is one series of cases in this grouping to which especial attention is directed and the group is presented with that purpose in view. I refer to the series of 123 cases of acute diffuse peritonitis in which operation was deferred. My view is that on the handling of these cases the question of the mortality rate largely rests. The whole question of mortality naturally hinges on the cases seen for the first time on the third or fourth day of the attack; cases with acute, spreading infection. It must surely have been genuine insight on the part of Ochsner, who laid bare the principle that one of the chief factors in the dissemination of the peritoneal infection was the vermicular movement of the small intestine. Since in acute appendicitis with peritonitis there is a constant regurgitation of the contents of the small intestine into the stomach, due to the closure of the inflamed ileocecal valve, the practise of gastric lavage, which empties not only the stomach but the small intestine as well, has its foundation in a rational conception of the diseased process. The basic principle of this treatment is that we thereby secure a condition, approximately at least, of physiologic rest to the inflamed area, which gives Nature the chance she has been seeking to complete the localization of the infection. Let me emphasize particularly the point that none of these patients was operated on immediately; they were all treated as outlined by Ochsner. With 2 exceptions, all were safely operated on at a later date, and in each case a gangrenous or ruptured appendix, with pus, was demonstrated at operation.

Contrast 85 cases of acute diffuse peritonitis operated on immediately with 7 deaths, or a mortality of 8.2 per cent, with 123

cases of acute diffuse peritonitis in which operation was deferred, with 2 deaths or a mortality of 1.6 per cent. Occasionally the Ochsner method has been supplemented by simple incision and drainage under local anesthesia, to relieve the absorption from pus under tension.

These 123 cases in the statistics are included in the group of 570 localized appendiceal abscesses; likewise the 2 deaths which occurred previous to operation are included in the 4 deaths in the same series. These cases have been separated for the purpose of illustrating the difference between the mortality when the cases of acute diffuse peritonitis were operated on immediately and when the operation was deferred.

Finally we will consider Group V.

	No. cases.	No. deaths.	Mortality.
Chronic appendicitis	1241	0	0
Acute appendicitis	688	1	0.15
Gangrenous, ruptured, localized abscess	570	4	0.7
Acute diffuse peritonitis	85	7	8.2
Cases in extremis; abscess drained, appendix not removed	9	3	33.33
Appendix removed in the course of other operations	366	1	0.27
	2959	16	0.54

In this group have been added 366 cases in which the appendix was removed in the course of other operations. Many times the appendix has been removed during the course of other operations, but there have only been included in these 366 cases those appendices in which evidence of inflammatory disease was unmistakable.

The group has this bearing on our study; it shows to what extent the removal of the appendix during the course of other operations increases the surgical risk. This group is a résumé of the entire work; it represents not only the best, but all that I have been able to accomplish in this field of endeavor. It also shows that the mortality over all in 2959 cases is represented by 16 deaths, or a mortality of 0.54 per cent.

In 1909, at the request of the late Dr. John Munroe, of Boston, I presented a paper on appendicitis before the American Medical Association, in which I took substantially the same position then that I am taking today as regards the management of the cases

of acute appendicitis complicated by the presence of diffuse peritonitis. I am more firmly established today than ever before in the belief that the secret of the mortality lies in the deferred operation as applicable to this particular group of cases. We must ever keep clearly in mind that in these cases we are handling appendicitis plus the consequent peritonitis.

If the thing for which contention is made is really a principle that should underlie the management of such conditions, then there should be a broader field of application than simply in relation to this type of appendicitis. The principle of deferred operation, broadly speaking, must be applicable in greater or less degree to the whole field of emergency surgery. This principle should underlie our management of the cases of acute infections of the gall-bladder, duodenal ulcer, ectopic pregnancy, and I would go to the point of saying, in the presence of known penetrating, perforating wounds of the abdomen, it is a mistake to operate on every case immediately they are seen. Some years ago I published a paper on penetrating gunshot wounds of the abdomen in which there were reported 27 cases with a mortality of 10 per cent. I am perfectly convinced that a number of these cases were saved by deliberately taking the necessary time to improve the patient's general condition before operating.

You cannot formulate a rule that will fit all cases; when one tries to do so, one becomes mentally stalemated. The heart of the surgical problem is to grasp the principle so clearly that the method adopted will best serve the needs of the individual case.

I have tried to be frank, explicit and without controversy in seeking to establish a certain basic principle of surgical practice, for surely out of the morass of medical mysticism there must be, in front of us somewhere, a straight path of solid ground on which we might walk safely.

DISCUSSION

Dr. John H. Jopson, Philadelphia:

I just want to say a few words about this subject because of my interest in it. A few years ago Dr. Pfeiffer and I presented a paper before the Association, of which the title was "The Limitations of the Ochsner Treatment in Certain Cases of Suppurative Peritonitis." That paper perhaps was justified at that time by reason of the diversity of opinion on this subject, which was very easily proven by a search of the literature, which showed a great diversity of opinion among the Fellows of this Association.

Taking the statistics from various hospitals in this country and abroad, including hospitals in which the services were under the direction of Fellows of this Association, we found that there was no uniformity of opinion as to the best time in which to operate for general peritonitis, and there was also, perhaps in consequence of this diversity of opinion, a very wide difference in mortality. It is true that this difference in mortality depended to some extent on a difference in definition of what we meant by general diffuse peritonitis; but that is a question which time will not permit us to discuss.

In general, as we divided them at that time, the policy of operation was that of the interventionist, the abstentionist and the opportunist, and by and large we ranged ourselves on the side of what Dr. Pfeiffer christened the opportunists, and tried to arrive at that state of mind which Dr. Guerry has very happily described, in which, while holding to a general principle of management as laid down by Dr. Ochsner, and which I am inclined to think is one of the greatest monuments to his memory, we at the same time tried to differentiate, as far as possible, between those cases in which the policy of abstention from immediate operation was apt to be followed by disaster and those cases in which abstention was followed by a great decrease in the mortality rate.

We also tried to make one distinction, and that was to recognize those cases where under a policy of abstention or a nonoperative policy, and usually outside of a hospital, there occurred the sudden acute disaster of a secondary rupture of a collection into a free portion of the peritoneal cavity. We tried to find a syndrome which would enable us to diagnose such a condition, and our subsequent experience has led us to believe that we at least measurably arrived at such an ability.

I think, of course, the mortality of such a method of treatment as we define by Ochsner's name should include those cases which die in the hospital under our management before the delayed operation is

instituted, because they might be included in the class of cases of peritonitis so difficult to define in which an immediate operation would be superior to a delayed operation.

Dr. Vernon C. David, Chicago, Ill.:

A few months ago I read a report from one of the German clinics on the subject of mortality in appendicitis in which the mortality rate in localized appendiceal abscesses approximated 20 per cent. That is a very great difference in statistical evidence from the paper which we have just listened to this afternoon, and I think the essayist is to be congratulated on his results.

However, I must say that in my own experience where a localized abscess has formed I have not always had a happy experience in simply draining the abscess. By that I mean when the operation is performed, if one goes through the free peritoneal cavity—no matter how well we have been able to wall off the free peritoneum—and then enters this abscessed cavity, in a number of instances there is a very severe reaction, and in a few instances death has followed such a procedure. We, therefore, have adopted the idea which is used in draining abscess of the lung and in subphrenic abscess, and do these cases in two stages. We open into the peritoneum, and if the free peritoneal cavity is not obliterated, namely, the abscess is not in contact with the parietal peritoneum, we stop at that point, pack a little gauze next to this abscessed cavity, and in forty-eight hours simply put a finger in it and open it with the free peritoneal cavity obliterated. This idea probably is very old. I never happened to see it described, and simply recite it for its worth.

Dr. Astley P. C. Ashhurst, Philadelphia:

Cases of appendicitis not requiring drainage are of very little statistical interest, as nearly all such patients recover; not one in my own hands has died. But I want to put on record my statistics of complicated cases of appendicitis, though I have comparatively few to report. There are two reasons why I have so few of such cases. The first reason is that for the past ten years I have not done myself very many emergency operations for appendicitis; I have two good associates who do nearly all my emergency work. The other reason is that I have made it a rule, to which there are few exceptions, to operate *immediately* on every patient with appendicitis; I am sure that in this way the number of cases requiring drainage has been kept low. Perhaps a third reason might be added, and that is the fact that fewer patients are sent into the hospital so late that complications have already developed: when I first began to operate, and was doing only emergency work, nearly

every patient required drainage; whereas now my associates and I find that in a majority of emergency operations the wound may be closed.

Up to May 1, 1926, I find I have operated with my own hands on 243 patients with appendicitis so far advanced as to require drainage (see table). I have divided the cases into (1) those patients who were operated on as soon as possible after admission to the hospital, and (2) those in whose cases operation was delayed because it was thought immediate operation would hasten death.

TWO HUNDRED AND FORTY-THREE COMPLICATED CASES OF APPENDICITIS (TO MAY 1, 1926)

OPERATION ON ADMISSION TO HOSPITAL

	Total.	Recovered.	Died.	Mortality, per cent.
Primary abscess	96	88	8	8.3
Gangrene	44	42	2	4.5
Diffuse peritonitis	68	57	11	16.1
	208	187	21	10.0

DELAYED OPERATION. CASES OF DIFFUSE PERITONITIS

	Total.	Recovered.	Died.	Mortality, per cent.
Died without operation	5	0	5	100.0
Abscess drained:				
Appendix not removed	20	13	7	35.0
Appendix removed	10	10	0	
	35	23	12	33.0
Total	243	210	33	13.5

1. OPERATION ON ADMISSION. These cases are subdivided further by the clinical diagnoses made at the time of operation into cases of:

Primary Abscess. I mean by this that the patient develops an abscess during the attack, but without having first passed through any state which can be recognized from the patient's own history or from the attending physician's report as diffuse peritonitis. The mortality in my cases is 8.3 per cent.

Gangrene. By this I mean gangrene of the appendix with peritoneal contamination sufficient to require drainage of the wound, but without a walled-off abscess. The mortality in my cases is 4.5 per cent. I think gangrene of the appendix deserves recognition as a clinical entity apart from primary abscess. But some cases of gangrene of the appendix are accompanied by so little peritoneal soiling that no drain-

age is required. No such cases are included in this series. I do not believe that *perforation* of the appendix, as such, deserves recognition as a clinical entity; if the perforation occurs into preformed adhesions the case is one of abscess or gangrene; if it does not occur into preformed adhesions it produces *diffuse peritonitis.*

Diffuse Peritonitis. This term I use purposely because it is noncommittal. It does not define how widespread the peritonitis is, because I do not know; at operation I do not find out. All I see at the operation is the appendix and base of the cecum; very occasionally some of the small bowel comes into view. On opening the peritoneum there is free, turbid fluid spread diffusely in the abdomen, not localized by adhesions. Usually the fluid first encountered is only seropus, but sometimes it is frank pus. There is more or less inflammatory lymph on the structures which are seen; there is pus in the pelvis or in the right flank or in both (where the cellular contents of the seropus have collected by gravity); but there are no localizing adhesions. And because no exploration is done beyond the regions mentioned, I do not know how widespread the peritonitis may be. But from the physical examination of the patient before operation, and because sometimes (after first sucking the pelvis and right flank dry, and then raising the head of the table) I have been able to suck more pus out of the pelvis, I surmise that the peritonitis is *diffuse* (that is, not localized), without being *general.* By the latter term I mean peritonitis which involves all or very nearly all of the peritoneal cavity—not only the pelvis and both flanks, but also the regions of the spleen, liver, stomach and beneath the diaphragm. On such patients I do not operate, because operation will only hasten death.

It is difficult to express in words any rule for determining before operation which are cases of diffuse and which of general peritonitis; each patient is a law unto himself. But usually, *so long as the degree of abdominal rigidity is greater than the distention*, the case may be classed in the former category, and in such cases I have not postponed operation. Many cases of early diffuse peritonitis, showing only excess of turbid fluid, will not require drainage after operation. Such cases are not included in this series. The mortality in my cases of diffuse peritonitis requiring drainage is 16 per cent. This is high, but I believe very few of the patients who died could have been saved by any other treatment.

2. Delayed Operation. If I thought a patient admitted with peritonitis was so ill that operation on admission would surely hasten death, then operation was delayed; and the patient was treated for peritonitis by the strictest code of the Ochsner *régime:* nothing but the stomach tube by mouth, instillation of fluids by rectum, etc., and morphin.

Of 35 such patients under my personal care, 5 died without operation, never reacting at all. These cases should be counted in any computation of mortality, because naturally it is the mortality of the disease, not only the operative death-rate, that is of interest. Twenty patients reacted so as to form one or more abscesses which could be recognized, and these abscesses were opened without attempting to remove the appendix: of these 20 patients 7 died, a mortality of 35 per cent. Ten patients reacted so completely that it was thought advisable to remove the appendix at the time the abscess was drained; none of these 10 patients died. So that among 35 patients considered too ill for operation on admission to the hospital there were 12 deaths, a mortality of 33 per cent, to be compared with a mortality of 10 per cent when prompt operation was thought wise. No one can claim infallible judgment, and no doubt errors of decision occurred in some cases. This total mortality of 13.5 per cent (33 deaths among 243 complicated cases of appendicitis) is to be compared with the absence of mortality in cases of acute appendicitis without the complications of abscess, gangrene or peritonitis. The life of every one of these 33 patients could have been saved if the family or the family's physician had insisted on immediate operation at the onset of the disease. This statement is not invalidated by the fact that many patients may have been treated at home with recovery: *to delay operation in acute appendicitis is to gamble with death.*

Perhaps I should add that a very few patients in whose cases operation was postponed because of widespread peritonitis recovered without forming any abscess at all; these cases are not included in this series (though to do so would reduce my mortality) because at operation no cause was found for drainage, and I have no means of knowing that the patients did certainly pass through an attack of peritonitis; there was *nothing* found at operation to prove the previous existence of widespread peritonitis.

Finally, may I make this remark: I think we ought to recognize that those surgeons who advocate delay in resorting to operation are quite likely to err by classing as *recoveries due to delay* cases in which the patients were never very seriously sick in the first place; cases, I mean, in which many surgeons (as I believe correctly) would operate promptly, and find that the wound could be closed without drainage, and there would never be any question of the patient's recovery. Only those surgeons who look inside the belly can know the true condition of affairs during the acute stages of the disease.

Dr. Guerry (in closing):

Evidently there is still misunderstanding in the minds of some as to the exact significance of the groupings of the cases; else it could

not be suggested that turbid fluid in the abdomen, a conservative process, was being called diffuse septic peritonitis. I have done my level best to express the matter so clearly that no one could misunderstand it. Evidently, I have failed. I shall make one final effort. Turn for a moment to the study as contained in Group 4.

A. Chronic Appendicitis. There can hardly be misunderstanding here, for I mean precisely what the word chronic implies and nothing more.

B. Acute Appendicitis. It seems to me that the text makes perfectly plain exactly what is meant by the group of acute appendix cases. Those of you who are sufficiently interested to read the text on this point cannot fail to understand.

C. Gangrenous, Ruptured, Localized Abscess Cases of Appendicitis. Under heading *C* of the study of the first group, we believe, one will find not only a frank description of this type of case, but so plainly stated that anyone who reads it must understand it.

D and *E* I will discuss together briefly, for these are the cases about which question has been suggested. There were 85 cases of acute diffuse peritonitis operated on during this stage of the disease. Let me be perfectly clear and emphatic on this point; they were not cases in which turbid fluid in the abdomen was mistaken for acute septic peritonitis. I mean precisely what the term acute diffuse peritonitis implies, nothing less and nothing more than simply that.

In section *E* of the same group there were 123 cases of acute diffuse peritonitis with deferred operation. The statement as to the character of cases treated in the 85 cases in Group *D* is applicable to the cases in Group *E*. It is quite true that the 123 cases in Group *E* were not operated on immediately that they were seen, but the text of the article makes absolutely plain that, while these cases were treated according to the principle of deferred operation, in each instance, when they were operated on, a gangrenous and ruptured appendix with pus was demonstrated at operation.

In conclusion, I would like to raise one final question. If there is not a definite, unmistakable tendency toward successful localization in cases of appendicitis complicated by the presence of pus, why is it that such a great number of these cases when seen for the first time, have already become completely localized?

THE EXPERIMENTAL PRODUCTION OF ABSCESS OF THE LUNG

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AND

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Abscess of the lung is a general term applied to a wide variety of suppurative conditions occurring within the lung. These conditions differ greatly in their mode of production, their morbid pathology and their response to therapy. They enter into a common category merely because they occupy the same organ, and they differ as widely as do comparable conditions within the liver, such as amœbic abscess and the abscess of an ascending pylephlebitis. Certainly the postpneumonic abscess, the bronchiectatic abscess and the postoperative abscess of the lung have a quite dissimilar etiology. All of these varieties undoubtedly belong in one or the other of the two great pathologic divisions of pulmonary suppuration, *i. e.*, endobronchial and parenchymatous suppuration. We feel, however, that further study of this subject from the point of view of morbid pathology is less likely to give a full appreciation of the condition than an attempt at experimental reproduction.

The studies reported here concern solely *postoperative abscess of the lung*. They form part of a general study of postoperative pulmonary complications and were undertaken in the hope that further proof might be found for the concept that a large proportion of such complications are due to embolism from the operative wound. We have[1,2] for ten years accepted the suggestion made in 1900 by Mikulicz[3] that embolism might be the cause of certain

so-called postoperative pneumonias. He was led to this assertion by the occurrence of such complications following operations under cocaine anesthesia. It is now generally accepted that, in addition to massive pulmonary embolism, certain of the other pulmonary complications such as pleurisy and pneumonia may have a similar etiology. Could we prove that postoperative abscess of the lung resulted from the same mechanism, considerable weight would be added to the explanation of these most serious operative sequelæ. Moreover, such a simplification, by bringing many complications within the limits of a single mechanism would forcibly indicate the lines along which these undesirable sequelæ might be avoided, since the blame for the complications would then rest squarely upon surgical technic and operative skill.

We have been able to assemble from the literature 1908 cases of abscess of the lung. Of these 29.6 per cent are postoperative, and 14.6 per cent, or approximately one-half, follow the operation of tonsillectomy. This frequency following tonsillectomy has resulted in the conception that abscess of the lung is a complication peculiar to this type of operation. Investigation does not substantiate this. The fact is that tonsillectomy is a very common operation and that abscess of the lung is a relatively infrequent postoperative complication. It follows the operation of tonsillectomy no more frequently than operations in any other septic field.

The apparent frequency of abscess of the lung following tonsillectomy was thought to be due to the special liability in this operation to aspiration of infected buccal content. It has long been known that during every general anesthetic mouth contents are aspirated into the lung,[4,5,6] and in the case of tonsillectomy, with the operative field close to the respiratory orifice, the danger seemed too obvious. Moreover, the reports of such complications from operators working upon patients in the upright position[7] seemed to further justify this feeling. As a result of such reasoning, considerable investigative work has been done attempting to reproduce a similar pulmonary suppuration in animals.[8,9,10] Many kinds of bacteria, plugs of meat and foreign materials have been introduced into the lung by insufflation, by the bronchoscope and by various ingenious methods calculated to lead the infected material into the finer ramifications of the bronchial tree. So far as we can determine, no one has been able to reproduce in animals typical abscess of the lung by such methods.

There is, moreover, another side to the problem. In the first place, postoperative pulmonary suppuration is not peculiar to the operation of tonsillectomy. Again this dire sequela follows upon tonsillectomy when the operation is performed under local anesthesia.[11,12] Further, the clinical history is not that of an immediate postoperative pulmonary upset. In fact, there frequently occurs a period of normal convalescence until, at a period seven to fourteen days postoperative, preceded by or synchronous with pleuritic pains, the symptoms of pulmonary disease commence and gradually are intensified. In addition there is the evidence that the endobronchial apparatus is well adapted to a defense against infection. Inspired foreign bodies rarely result in true pulmonary suppuration, though there may occur endobronchial irritation, infection and subsequent dilatation of that part of the air passages lodging the foreign body.[13] As further evidence of the great defensive mechanism within the bronchi, may we cite the following case:

E. T., aged forty-three years, complained of dysphagia. The diagnosis of carcinoma of the esophagus was made by direct visualization December 4, 1924. He was treated by the implantation of radium seeds. At the patient's request, gastrostomy was performed September 30, 1925. March 18, 1926 the patient commenced regurgitating by mouth food given *via* the stomach catheter. This was accompanied by paroxysms of coughing. This condition continued until his death April 29, 1926. Autopsy showed complete stenosis of the esophagus and a fistula between the esophagus just below this point and the left primary bronchus; the left lung was clear; there was pneumonia in the right lower and middle lobes.

It seems established in this case that for at least five weeks the patient had a communication between his stomach and his left lung, and yet the left lung was unaffected!

We had long felt that evidence of this nature was sufficient to justify grave suspicion of the conception that aspiration was the cause of pulmonary suppuration. Knowing of the failure of the experimental work in which attempts have been made to reproduce abscess of the lung by the instillation of infected materials into the air passages, it occurred to us that postoperative abscess of the lung might well be of embolic origin as are many other postoperative complications.

Before attempting to produce abscess of the lung by the use of

infected emboli and in order to free ourselves of criticism by those who still hold to the theory that aspiration gives rise to this condition, we performed a considerable number of experiments in which we placed infected materials with a bronchoscope into the finer ramifications of the bronchial tree. We used pieces of infected meat, infected tonsil, peanuts, etc. In no one of the seventeen attempts could we reproduce typical abscess of the lung.

FIG. 1.—Segment of femoral vein tied at base and held open for the insertion of a piece of lead and the bacterial emulsion.

This was not surprising to us because other investigators had failed previously in similar attempts.

A METHOD FOR THE PRODUCTION OF ABSCESS OF THE LUNG.[14] We then attempted the production of abscess of the lung by the instillation of septic emboli. Dogs were used in these experiments, and once the procedure became standardized we were able to produce abscesses in 100 per cent of our animals. During the early experiments we inserted into the jugular vein of the animals pieces of infected tonsil, infected meat, etc., and though we achieved some abscesses, it was frequently the result that an

extensive pneumonitis of the lobe in which the embolus lodged was produced. This rapidly broke down and often perforated into the pleural cavity resulting in death. We felt that, although it was necessary to have the correct number and type of organisms present, it was equally necessary that some sort of local immunity be produced in the pulmonary tissue before the majority of the organisms were set free. It seemed to us simpler to set up a temporary artificial barrier about the infected embolus than to raise beforehand the defense reaction in the pulmonary field. The

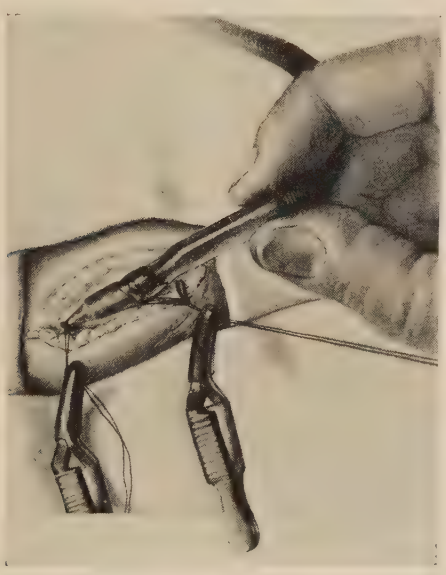

FIG. 2.—Segment of vein in glass cannula filled with salt solution about to be forced into jugular vein.

procedure, which finally became standardized as the most satisfactory for the production of abscess of the lung, consisted in constructing an embolus of a segment of vein (Fig. 1), which was filled with a culture of the desired organisms. We excised a small segment of the femoral vein, ligated one end, filled this capsule with the organisms and added a piece of lead filing coated with paraffin to render it inert. This bit of metal enabled us to recognize in immediate roentgenograms the site where the embolus lodged. After tying off the other end of this small capsule it was set free in the jugular or femoral vein (Fig. 2). In over 60 per cent

of our experiments the embolus lodged in the left lower lobe, due, we believe, to the straighter course of the vessel and the greater volume of blood going to this lobe. This course was taken, therefore, for the same reason that directs the lodgment of emboli in human cases where the majority go to the right lower lobe.

The following experiment is typical of the series, and serves to illustrate the simplicity of the procedure:

Experiment I.—Dog Y 38 (weight, 12.1 kg.), November 11, 1925, was given morphine, gr. $\frac{1}{4}$. Under ether anesthesia, a segment was removed from the right femoral vein, inoculated with cultures of Staphylococcus aureus, Bacillus coli and pneumococcus, Type II, and introduced into the left jugular vein. A roentgenogram localized the foreign body in the left lower lobe (Fig. 3).

November 13: According to the roentgenogram there was beginning infiltration about the foreign body (Fig. 4). The animal ate his food well.

November 16: A roentgenogram of the chest showed a definite abscess cavity in the left lower lobe where a clear zone containing the bit of lead appeared in the center of the infiltrated area (Fig. 5). The animal ate only a small amount of food.

November 19: The abscess cavity in the left lower lobe was larger. The surrounding infiltration of the lung remained about the same (Fig. 6).

November 27: Subsequent roentgenograms of the chest had shown a subsidence of the infiltration and the abscess cavity had decreased considerably in size. The left lower lobe was removed by operation. In the removed lobe an area of induration could be felt near the tip of the lobe. The overlying visceral pleura was considerably thickened. On section, thick indurated tissue was encountered within which a small abscess cavity was still present (Fig. 7). Microscopically, the lining of the cavity was composed chiefly of fibrous tissue. Considerable cell exudation was still present.

Discussion. By this method of freeing infected emboli into the venous circuit, we have been able to produce true abscess of the lung. Such an abscess starts as a parenchymatous lesion. Its establishment depends undoubtedly upon many factors. Under the circumstances imposed by us in our experiments, the type of organism present and the physical property of the covering of the embolus seem to be of some importance. We found that freeing a simple infected and *uncovered* clot into the venous circuit usually

FIG. 3.—Roentgenogram of Dog Y 38. Experiment I, immediately after embolism. The piece of lead in the left lower lobe indicates the position of the embolus.

FIG. 4.—Roentgenogram of Dog Y 38. Experiment I, two days after embolism. There is beginning infiltration about the foreign body.

FIG. 5.—Roentgenogram of Dog Y 38. Experiment I, five days after embolism. A well-defined abscess cavity is present.

FIG. 6.—Roentgenogram of Dog Y 38. Experiment I, eight days after embolism. The abscess in the left lower lobe is distinct. The lead fragment lies in a clear central zone.

resulted in a general pneumonitis rather than a walled-off abscess. It would seem as if the covering of the venous segment permitted the establishment of a walling-off process before the full effect of all the organisms in the capsule was produced. Or the influence of the venous segment as an organic foreign body may be of importance. Possibly this means that the actual physical makeup of any single infected embolus may play a dominant rôle as to whether abscess of the lung is or is not to result. It is conceivable that clots of variable structure and infectivity may be set free from

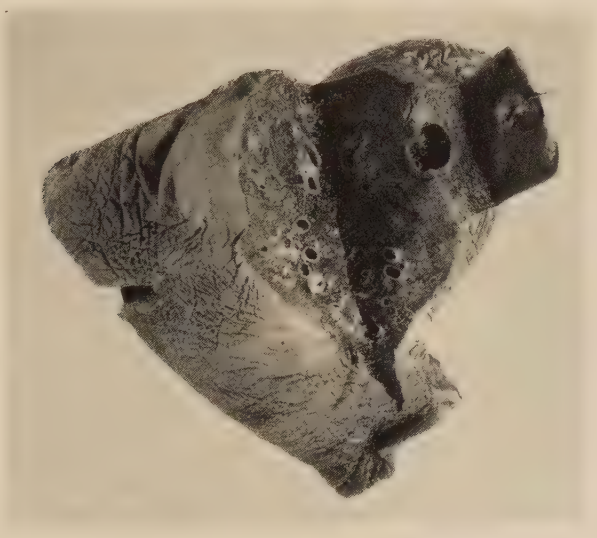

FIG. 7.—Left lower lobe removed from Y 38. Experiment I, sixteen days after embolism. A small abscess is still present.

the operative field. Those clots, in which the majority of the organisms are centrally located, and in which the clot has an unusually tough peripheral coat, should be more likely to cause an abscess.

It would seem, however, that such an explanation could apply to only a limited number of actual cases. This necessitated further study concerning the mechanism by which local tissue immunity might be produced. It was apparent that there must be present some factor which tended to keep the process well localized. Unless a clot was so constructed that it had a tough outer coat, that factor must deal with local tissue immunity. We felt that, if

we could raise the local immunity of an animal by vaccination with an organism to be used later in an infected thrombus, we might well establish a high local immunity which would restrain spreading of organisms from the local field, bring about an intense local resistance, and thus create an abscess. Moreover, as post-operative abscess of the lung occurs in cases already infected and thus already immunized, it would appear that such experiments would more nearly reduplicate what actually occurs in human cases. The following protocols of experimental and control animals demonstrate the proof of these contentions.

Experimental Studies of the Rôle of Local Immunity in the Production of Abscess of the Lung. Four animals were comprised in each experiment. One animal was used as a normal control, one was immunized by injecting an avirulent strain of B. coli intravenously, one animal had small sterile emboli (starch granules) set free in the jugular vein in an attempt to see if non-infectious material would also create high local resistance, and the fourth animal was submitted to an abdominal operation (lateral intestinal anastomosis) in an effort to see whether such a procedure would bring about a definite general immune reaction sufficient to create local pulmonary resistance and thus wall off the subsequent pulmonary infection. We felt that we covered the major possibilities for the establishment of a high local resistance in the lung with the normal control, the preoperative aseptic embolic control and the simple operative control animals. When these animals had been sufficiently prepared they were submitted to embolism.

The embolus was prepared as follows: A virulent strain* of B. coli was obtained, grown on an agar slant, and the culture washed off with salt solution and thoroughly shaken; blood was drawn under aseptic precautions, the suspension of B. coli at once added and the tube thoroughly shaken to ensure equal mixing before clotting. The whole was then allowed to clot and eighteen hours later the clot was carefully divided into four equal parts and these clots slipped into the jugular vein of all four animals. Each fragment of clot was roughly 10 x 4 mm. in size.

* Secured from Dr. B. Steinberg of the Department of Pathology, Western Reserve University. This organism was of sufficient virulence to kill a dog in six hours by the intraperitoneal injection of one washed agar slant mixed with gum tragacanth.

The experimental work dealing with this phase of the question is not sufficiently complete to permit us at this time to give final reports and full proof of our ideas regarding the rôle of local tissue immunity and the method of its production. We have, however, sufficient experimental evidence to strongly indicate that the establishment of such a local immunity plays a dominant part in the production of abscess of the lung using the method as described.

Before reporting such experiments may we repeat the following facts: 1, Simple infected clots set free in the jugular vein of dogs usually give rise to a diffuse pneumonitis which will result in fatality or recovery according to the virulence of the organism; 2, an infected embolus enclosed in a capsule (segment of vein) will result in an abscess of the lung.

Experiment A. Dog I (normal control), weight 16.9 kg., May 15, 1926, was given morphine, gr. $\frac{1}{4}$. Under novocaine anesthesia the left jugular vein was exposed, opened and the eighteen-hour clot infected with the virulent Bacillus coli organism was introduced.

May 16: Twenty-four hours after the injection of the clot the dog appeared ill and would not eat. The respiratory rate was so rapid that an *x*-ray plate of the chest could not be taken. It was noticed that at times the dog would cough and bring up a frothy, bloody sputum. The rectal temperature was 41.3° C.

May 17: The dog died thirty-six hours after the injection of the infected clot.

Necropsy. There was considerable cloudy hemorrhagic fluid in the left pleural cavity. The lungs showed some congestion at the right lower base; otherwise the lobes on the right side were air-containing and apparently normal. The left upper lobe showed some congestion. The left lower lobe was quite densely consolidated (Fig. 8). On section the cut surface of this lobe was of a deep reddish color (red hepatization stage of pneumonia).

Dog II (bacterial immunized control), weight 8 kg., May 15, 1926, was given morphine, gr. $\frac{1}{4}$. An attempt had been made to immunize this animal by injecting intravenously a platinum loopful of avirulent Bacillus coli organisms suspended in 10 cc. of salt solution. Three injections at intervals of two days had been carried out ten days previously. The left jugular vein was exposed under novocaine anesthesia, opened and the eighteen-hour clot infected with the virulent Bacillus coli organism was introduced.

May 16: The dog ate most of his food and did not appear ill although

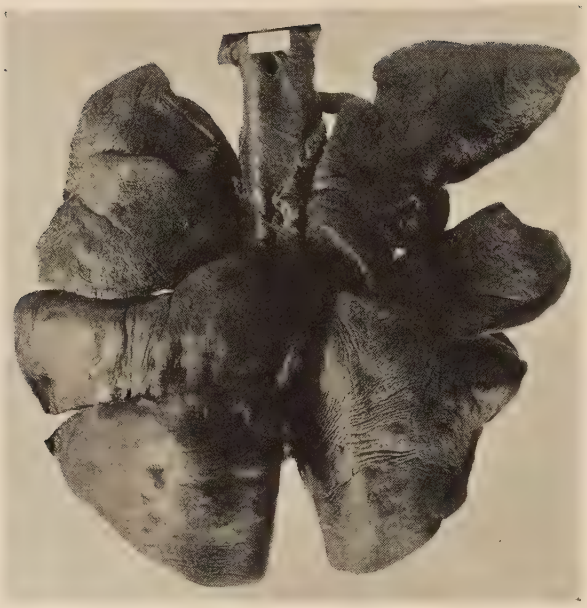

FIG. 8.—Lungs removed at necropsy from Dog I (normal control) thirty-six hours after the intravenous injection of a clot infected with virulent Bacillus coli organisms. The left lower lobe is quite densely consolidated.

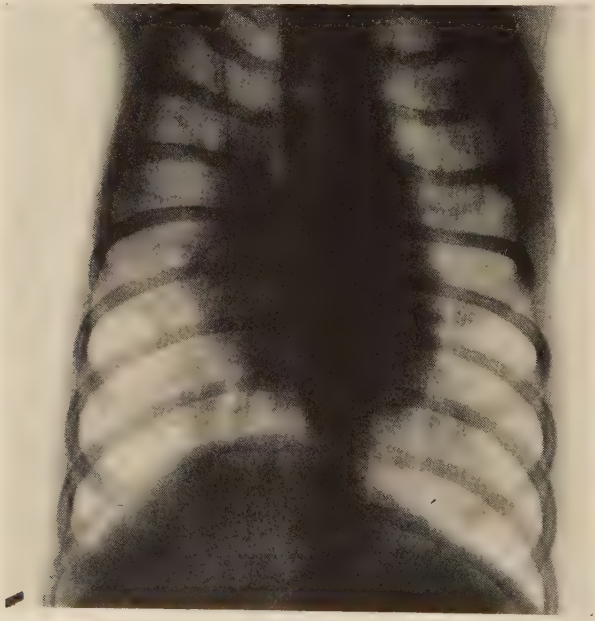

FIG. 9.—Roentgenogram of Dog II fifty-four hours after the intravenous injection of a clot infected with virulent Bacillus coli organisms. There is a beginning consolidation of the right lower lobe.

his temperature was 39.7° C. A roentgenogram of the chest showed normal lung fields.

May 17: The dog ate his food. The rectal temperature was 39.9° C. A roentgenogram of the chest was taken in the morning. The lung fields appeared quite normal. Another roentgenogram of the chest was taken in the late afternoon and showed a beginning area of cloudiness in the right lower lobe (Fig. 9).

May 18: When seen about 9 A.M. the dog was in extremis. The respiratory rate was very rapid and the rectal temperature was 40° C. A roentgenogram of the chest just preceding death showed a dense cloudiness of the entire right lower lung (Fig. 10).

Necropsy. There was a slight amount of purulent hemorrhagic fluid in the right pleural cavity. The left lung appeared normal. The right upper and middle lobes showed some congestion but were air-containing. The right lower lobe was densely consolidated and cut with resistance (Fig. 11). The cut surface of this lobe presented a deep reddish color. There was no localized abscess present. The animal had apparently established some degree of immunity which was, however, not sufficient to prevent the diffuse lower lobe infection. It is interesting to note, however, that this animal, a smaller animal than the normal control, was able to withstand the infection for a greater period of time.

Dog III (preoperative aseptic embolic control), weight 10 kg. May 15, 1926, was given morphine, gr. $\frac{1}{4}$. This animal had had three intravenous injections at intervals of two days of 10 cc. of starch solution ten days previously. Under novocaine anesthesia the left jugular vein was exposed, opened, and the eighteen-hour clot infected with the virulent Bacillus coli organism was introduced.

May 16: The animal died about eighteen hours following the injection.

Necropsy. There was a slight amount of cloudy hemorrhagic fluid in the right pleural cavity. The left lower lobe showed some congestion but otherwise the left lung was air-containing. The right upper and middle lobes showed a moderate degree of congestion. The right lower lobe was densely consolidated and there was a slight amount of exudate on the visceral pleura (Fig. 12). On cut section the lobe showed numerous grayish patches, but a reddish color was predominant.

Dog IV (simple operative control), weight 16.9 kg., May 15, 1926, was given morphine, gr. $\frac{1}{4}$. This animal had been submitted to an abdominal operation (lateral intestinal anastomosis) two days previously. Under novocaine anesthesia the left jugular vein was exposed, opened and the eighteen-hour clot infected with the virulent Bacillus coli organism was introduced.

FIG. 10.—Roentgenogram of Dog II, seventy-two hours after the intravenous injection of a clot infected with virulent Bacillus coli organisms. The right lower lobe is densely consolidated.

FIG. 11.—Lungs removed at necropsy from Dog II, seventy-two hours after the intravenous injection of a clot infected with virulent Bacillus coli organisms. The right lower lobe is densely consolidated.

FIG. 12.—Lungs removed at necropsy from Dog III, eighteen hours after the intravenous injection of a clot infected with virulent Bacillus coli organisms. The right lower lobe is consolidated. The right upper and middle lobes and the left lower lobe show some congestion.

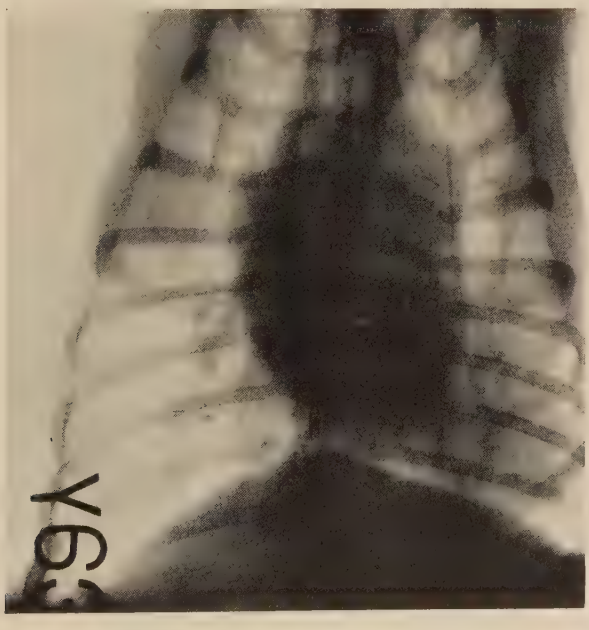

FIG. 13.—Roentgenogram of Dog IV, forty-eight hours after the intravenous injection of a clot infected with virulent Bacillus coli organisms. The left lower lobe shows a diffuse cloudiness.

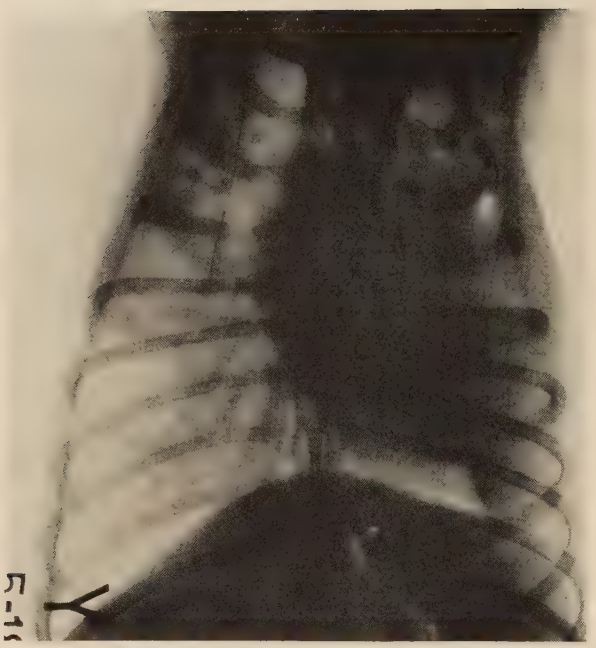

FIG. 14.—Roentgenogram of Dog IV, seventy-two hours after the intravenous injection of a clot infected with virulent Bacillus coli organisms. The left lower lung casts a dense shadow.

FIG. 15.—Roentgenogram of Dog IV, ninety-six hours after the intravenous injection of a clot infected with virulent Bacillus coli organisms. The left lower lobe shows less infiltration with areas of lessened density.

Fig. 16.—Left lower lobe removed at operation from Dog IV, ninety-six hours after the intravenous injection of a clot infected with virulent Bacillus coli organisms. The lobe is quite solid and numerous areas of exudate present on the visceral pleura beneath which are areas of softening.

Fig. 17.—Appearance of the lobe removed from Dog IV, after sectioning. One fairly large abscess can be seen near the hilus.

May 16: The dog appeared somewhat ill but ate part of his food. The rectal temperature was 40.5° C. A roentgenogram of the chest showed an indefinite area of cloudiness in the left lower lobe.

May 17: The animal ate a part of his food. The respiratory rate was definitely increased and it was noticed that the dog coughed occasionally. The rectal temperature was 40.7° C. A roentgenogram of the chest showed a definite area of mottled cloudiness in the left lower lobe (Fig. 13). A roentgenogram taken late in the afternoon showed an even greater area of cloudiness in the left lower lobe.

May 18: The animal continued ill but ate some food. The rectal temperature was 38.9° C. A roentgenogram of the chest showed an even greater area of consolidation of the left lung (Fig. 14).

May 19: The animal's general condition seemed improved. The rectal temperature was 40° C. A roentgenogram of the chest showed the area of cloudiness in the left lower lung to be less dense, but there were numerous areas of lessened density suggestive of abscess formation (Fig. 15). It was decided to remove the left lower lobe by operation. At operation numerous friable adhesions plastered the left lower lobe to the lateral chest wall and to the adjoining upper lobe. These were easily broken down and the lobe was removed. The removed lobe was consolidated throughout except for a small area near the tip which was air-containing. The lobe was of a dark red color and presented numerous areas of grayish exudate on the visceral pleura beneath which areas of softening could be felt. Near the hilus of the lobe there were several areas of broken down lung tissue (Fig. 16). The lobe was sectioned and several small abscesses were encountered containing thick whitish pus. Near the hilus of the lobe a fairly large abscess cavity was encountered approximately 1 cm. by 3 cm. (Fig. 17).

Discussion. The foregoing experiment demonstrates facts that have been corroborated repeatedly in this laboratory. In the first place virulent infected clots set free in the venous circuit result in fatal pneumonitis. Histologic examination shows a process identical with true lobar pneumonia. In the second place immunization with avirulent organisms or by previously operating upon the animal in a field where organisms are already present (the intestines) yields a varying degree of protection. Both Dog II (bacterial immunized control) and Dog IV (operative control) outlived Dog I (the normal control animal). Aseptic emboli seem to instigate insufficient immunity to affect the outcome (Dog. III). In this particular experiment, the normal control animal (Dog I) survived eighteen hours, the bacterial immunized control animal

(Dog II) survived seventy-two hours and the operative control animal (Dog IV) was apparently recovering when lobectomy was performed. Abscess of the lung resulted only in Dog IV. We feel that this indicates that abscess results when immunity has been sufficiently stimulated. The fact that Dog IV outlived Dog II indicates that immunity reactions were more highly stimulated.

These experiments will be elaborated upon and repeated but even the single group reported would seem to indicate that in the establishment of a postoperative abscess of the lung the two factors of *embolism from the wound* and *the local immune reactions in the lung* play dominant rôles. The type of organism present, the physical character of the clot, immunity established by previous infection with similar organisms, the presence of organic matter other than simple clot in the embolus, and the number and virulence of both pathogenic and saprophytic organisms within the bronchial passages themselves may play a variable part.

Why such lesions in dogs tend to heal within two weeks unless they kill in the first few days seems to be explained (1) by the horizontal plane of the dog's bronchial tree enabling freer drainage than in man and (2) by the fact that animals have because of more frequent exposure a far higher resistance to all infection.

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DISCUSSION

Dr. George P. Müller, Philadelphia:

I believe that this paper of Dr. Cutler is exceedingly valuable in that it helps us to get rid of certain preconceived ideas. There is no reason why we should be so insistent upon the aspiration theory of lung abscess. I remember that, years ago, when Dr. Ellsberg devised his apparatus for intertracheal anesthesia, I became enthusiastic about it and built a similar apparatus. For a number of years I used endotracheal anesthesia, not only for operations upon the mouth, but for all kinds of operations. Dr. Frazier used the apparatus many times until rather recently in cases of spinal-cord tumor where the patient was recumbent. These amounted to several hundred; yet in none of them did lung abscess develop. Occasionally the machine would stop, something would happen to the electricity, and you could easily conceive that material could then be blown into the lung upon the reëstablishment of the pressure current. More recently, lipiodol and bismuth have been injected by the bronchoscopists, and yet we have not so far heard of the frequent development of lung abscess following the procedure.

Several years ago, at the University of Pennsylvania, Fetterolf and Fox showed that septic emboli could be readily carried to the lung from a septic throat and set up a lung abscess. Fetterolf regarded this as the principal cause of abscess of the lung after tonsillectomy, believing that the frequency of abscess of the lung that has seemingly occurred recently could be traced to the development of what is called the modern operation, that is, not the simple tonsillectomy with a tonsillotome, but the digging out of the tonsil from the depths of the throat in the enucleation method, exposing raw areas and twisting veins, and then, if infection of these clotted veins occurred, that emboli could be carried into the lung. Fetterolf told me that he had noticed frequently in children a period, four or five days after operation, when they would have some cough and bloody sputum and that was all. He believed those were aseptic emboli, and that, in an occasional case, where the resistance of the lung was not sufficient or the infection still greater, abscess would develop.

Dr. Edward W. Archibald, Montreal, Canada:

The theory of the causation of lung abscess by septic emboli is very attractive and very plausible, and I think we all believe that that is what occurs in a great many instances. I would not like, however, that our older theory of aspiration pneumonia followed by abscess should be entirely discarded.

I would call attention, in this connection, to experiments of the last few years in the matter of tuberculous infection. For instance, Dr. Soper, at Saranac Lake, repeated experiments of others (whose names I forget) of three or four years ago, whereby 1 cc. of a broth suspension of tubercle bacilli was placed inside of the nostril of a rabbit, the rabbit being under deep anesthesia; after which, the animal being laid upon one side or the other, there would develop regularly a massive tuberculous pneumonia or bronchopneumonia on the side on which the rabbit lay.

If mere aspiration can cause a tuberculous lung infection, can it not equally be the means of causing any kind of lung infection? And if so, why not of abscess formation? With varying types of organisms, one may assume that in one case the inflammation will go on to resolution, while in another case it will form an abscess.

Lipiodol injections into the bronchial tree, which we have recently been doing, show that during cough the lipiodol is spread into the alveolar spaces very freely. We know, also, that particles of carbon by mere force of inspiration can be carried into the peripheral alveoli. One might ask, if that is the case, why do not abscesses develop more frequently from aspiration? I believe the result is largely dependent upon local conditions, as to whether the contents of the alveoli—aspirated matter, carbon particles and so on—can get out easily into the smaller bronchioles. It depends a good deal upon the force of expectoration and of cough. Obviously there are several other factors, such as prolonged anesthesia, length of stay of the foreign material in the alveoli, local trauma, virulence of organisms and so on.

I merely want to claim for the older aspiration theory a measure of support.

Dr. Cutler (in closing):

I am very grateful to Dr. Müller for bringing up the work of Fetterolf and Fox, because it was a very important contribution. Some years ago I had a long correspondence with these authors as the result of a statement, in an article on postoperative pulmonary complications, that there were cases of abscess of the lung which occurred after tonsillectomy under local anesthesia. These investigators did some excellent work on the actual pathologic changes that take place in the bed of a tonsil after operation, and they showed that extensive thrombosis, from which embolism might well occur, is often present.

The whole case of the aspiration theory cannot be properly covered. I agree that one can produce infection in the lung if one injects a great number of bacteria. Such an experiment bears no relation, however, to what could occur in human cases. In our experiments we attempted

to more nearly simulate the possibilities in man. The number of bacteria that we place in the lung through a small piece of infected clot is not at all comparable to placing a large fragment of a fresh culture in the nose of an animal. The latter results in an overwhelming infection. With such a dosage one can, of course, produce in the laboratory almost anything. If, however, one attempts to simulate what occurs under human conditions, and uses a small dosage, then one must and can study not only the effect of the injected material *per se*, but the reaction of this material. This makes the study somewhat more complicated, but certainly should result in a fuller appreciation of the reaction in man.

We feel that it is this process of reaction and the existence of stimuli, previous to the setting free of an infected clot, which increase the local immunity and which dictate the pathologic lesion that is to result. If there is a high immunity the lesion will rapidly become walled off and an abscess will result.

LATE RESULTS AFTER AMPUTATION OF THE BREAST FOR CARCINOMA

By ALEXIS V. MOSCHCOWITZ, M.D.

AND

RALPH COLP, M.D., AND PERCY KLINGENSTEIN, M.D.
(By Invitation)
NEW YORK CITY

THANKS to the efforts of Halstead and Willy Meyer, the modern operative treatment of cancer of the breast may be said to have become standardized. Collective statistics bear proof that the number of patients alive and free from recognized carcinoma, from three to five years after operation, has increased measurably, as a result of the more radical and anatomically perfect operation. In 1924, Lane-Claypon, in an analysis of 20,000 cases of operated breast cancer collected from the literature of the world, tabulated the following results based upon a three-year period of apparent cure: Of 7029 patients operated upon, so to say incompletely, *i. e.*, before the advent of the Halstead-Meyer operation, 29.2 per cent were free from recurrence after a three-year interval. On the other hand, of 8921 patients undergoing the complete operation, 43.2 per cent were free from recurrence after a similar period of time. The reported series of Judd and Sistrunk, Sistrunk, Greenough and Simmons, Primrose, Ochsner and Cabot, in this country, bear further evidence of the improvement in the operative results. I, too, shared in the general optimism pervading the literature, particularly in respect to the early cases of breast carcinoma. It was indeed a great surprise to me, therefore, that in 1924 B. J. Lee, in an admirable paper read before this Association, recounted the results obtained in the operative treatment of 87 primary operable cases of breast carcinoma, treated at the New York Hospital, all of whom have been followed for a

period of at least five years, and reported only 15 per cent to be alive and free from tangible recurrence. At that time, I determined, if possible, to ascertain the results of our efforts in the treatment of breast cancer at Mt. Sinai Hospital,* and it is the results of this investigation with a few conclusions drawn therefrom that I wish to submit for your consideration.

For this purpose we have reviewed the cases of breast carcinoma admitted to the wards and private pavilion of Mt. Sinai Hospital from January 1, 1915, to December 31, 1924. In all, 374 patients were discharged with a clinical diagnosis of carcinoma of the breast, exclusive of those patients who were readmitted for the treatment of local recurrences or metastases. To be sure, we have been unable to use this entire material. Particularly is this true of patients treated in the private pavilion of the hospital. Regrettably, we found here a great number of pathologic reports missing, and no case in this entire series is considered either from a statistical standpoint or is included in the follow-up report unless a pathologic report, unequivocally carcinoma, was affixed to the record. In a small number of cases the pathologic report upon a patient's first admission to the hospital was lacking; but if subsequent readmission for recurrence proved the excised specimen to be carcinoma, this was deemed sufficient proof that the original tumor was a cancer, and to permit inclusion of the case both in the follow-up report and statistical data. A number of patients (these will be considered as a separate group in a subsequent paper) were discharged without operation. In some cases operation was refused by the patient; in others the presence of supraclavicular or more distant metastases, adherence of the tumor to the chest wall, or contraindications apart from the local lesion, such as diabetes, pulmonary tuberculosis, etc., actuated the surgeon to withhold operative measures and to rely upon physical agents (x-ray or radium) as a theraputic measure.

This leaves for analysis 218 cases of proven breast carcinoma. Of this number we have been able to follow 139 cases: 14 were followed for a period of eleven years; 26 for a period of at least ten years; 31 for nine years; 37 for eight years; 56 for seven years; 73 for six years; 89 for five years; 115 for four years; 125 for three years and 139 for two years. The only explanation for the

* I take this opportunity to thank all my colleagues on the staff for permission to incorporate their cases in this contribution.

paucity in follow-up results is the fact that not all of the private records were open to us. A noteworthy fact, in spite of the difficulties encountered in following patients in a large metropolis, is that we were able to trace over 85 per cent of our ward patients, this in face of a not altogether adequate follow-up system prior to 1922.

All of these 139 patients were considered to be primarily operable and were subject to the radical operation. We realize that anything short of a five-year follow-up is entirely inadequate, and we will stress particularly the 89 patients whom we have been able to follow for at least this length of time. This statement, we believe, should be emphasized, because in this small series 14 cases have died of recurrent carcinoma* after a period of five years of apparent well-being. In parenthesis, we may add that we have abandoned the terms "cure" and "end-results," and have substituted the less-incriminating phrases, "free from recurrence" and "late results." All follow-up data are based upon information secured prior to January 1, 1926. Since that time we have learned of 2 deaths in this series. These are indicated in chart. We have deemed it advisable to classify all known recurrences as deaths from carcnoma, even though the patient was still living when last heard from. We feel that in these instances the patient is still suffering from carcinoma, and that the primary operative objective, namely, the eradication of the disease, has been defeated.

Sex In this series there were 214 females and 4 males, an incidence of 1.8 per cent of carcinoma of the breast in the male, which corresponds with the findings recently published by Judd and Morse. Of the 3 patients of whom we have follow-up notes, 2 are dead of recurrences at the end of one and three years, and 1 is living free from any evidence of carcinoma over a period of four years.

Age. Of the 218 cases, 12 were between twenty-one and thirty years of age; 59, between thirty-one and forty; 67, between forty-one and fifty; 46, between fifty-one and sixty; 32, between sixty-

* As a general rule we make the very important differentiation between recurrences and metastases. In view of the fact, however, that most of the cases that died did not die under our personal supervision, but were followed by the social service department, we have grouped both the recurrences and metastases together.

CHART I

Year.	Number of patients.	1 year.			2 years.			3 years.			4 years.			5 years.			6 years.			7 years.			8 years.			9 years.			10 years.			11 years.		
		Died.	Recurrences.	Living.	Died.	Recurrences.	Living.	Died.	Recurrences.	Living.	Died.	Recurrences.	Living.	Died.	Recurrences.	Living.	Died.	Recurrences.	Living.	Died.	Recurrences.	Living.	Died.	Recurrences.	Living.	Died.	Recurrences.	Living.	Died.	Recurrences.	Living.	Died.	Recurrences.	Living.
1915 .	14	0	4	10	4	1	5	0	1	4	0	0	4	0	0	4	0	0	4	1	0	3	0	0	3	1	0	2	1	0	1	0	0	1*
1916 .	12	1	0	11	6	0	5	0	0	5	2	0	3	0	0	3	0	0	3	0	0	3	1	1	1	1	0	0	0	0	0†			
1917 .	5	1	0	4	0	0	4	0	0	4	2	0	2	1	0	1	0	0	1	0	0	1	1	0	0	0	0	0						
1918 .	6	0	0	6	0	1	5	1	0	4	1	0	3	0	0	3	1	0	2	0	0	2	0	0	2									
1919 .	19	1	0	18	3	0	15	2	0	13	2	0	11	1	0	10	3	0	7	0	1	6‡												
1920 .	17	3	0	14	1	0	13	2	0	11	1	1	9	1	0	8	2	0	6															
1921 .	16	3	0	13	3	0	10	4	0	6	2	0	4	2	0	2§																		
1922 .	26	6	0	20	1	0	19	2	0	17	4	1	12																					
1923 .	10	4	0	6	3	0	3	1	0	2																								
1924 .	14	2	1	11	2	2	7																											
Total .	139	21	5	113	23	4	86	12	1	66	14	2	48	5	0	31	6	0	23	1	1	15	2	1	6	2	0	2	1	0	1	0	0	1
Number of cases followed	. . .	139			139			125			115			89			73			56			37			31			26			14		

* Patient died February, 1926 from intercurrent disease.
‡ One patient died March, 1926, from carcinosis.

† One patient living with a recurrence.
§ One patient died March, 1926, from carcinosis.

one and seventy; 1, between seventy-one and eighty. In 1 case, the age was not mentioned.

TABLE I

Age periods.	Number of cases.	Percentage.
21 to 30	12	5.5
31 to 40	59	27.0
41 to 50	67	30.0
51 to 60	46	21.0
61 to 70	32	14.5
71 to 80	1	0.5
Not mentioned	1	0.5

It is almost impossible to evaluate the rôle of age in longevity; the presence of involved axillary glands, extent of the disease, etc., make the comparison of one factor as opposed to the other almost impossible. Of the 89 cases which we have followed for at least a five-year period, 6 were operated upon before the age of thirty years. All of these cases have since died of carcinoma. The average duration of life after operation was two years. Of the patients who presented themselves for operation in the fourth or fifth decade of life, the average length of life after operation was three and a half years. In this group we count 13 patients who are at present living and free from recurrences. The average length of life in those patients operated upon after the sixtieth year was four and two-tenth years. Two of these patients are alive and well. It would seem, other factors being constant, that carcinoma of the breast in the young has a more malignant tendency, this impression corresponding to the well-known very malignant and rapid course of carcinoma of the cervix and rectum in the third decade of life.

As it may be of interest to show the relationship of the pathologic glandular involvements in the various age groups, we append Table II, which shows this relationship in 56 patients who died in less than three years after operation.

TABLE II

Ages.	Number of cases.	Glandular involvement.	Glands not involved.	Not mentioned.
21 to 30	6	5	0	1
31 to 40	13	11	2	0
41 to 50	17	10	4	3
51 to 60	12	7	2	3
61 to 70	7	2	3	2

Trauma as an Etiologic Factor in Breast Cancer. In 78 cases of this series trauma was specifically denied by the patient upon interrogation; 12 patients gave a history of a trauma, the time interval between the trauma and the appearance of a noticeable mass in the breast varied between six weeks and twenty-four years. The average time which elapsed was eight months.

Preceding Breast Abscess. Ten patients gave a history of a preceding mammary abscess. The time interval between the infection and the appearance of a mammary tumor was ten years. In 1 case a breast abscess was incised four weeks prior to admission to the hospital, but in this case it seems more than likely that the condition was an inflammatory carcinoma with necrosis. The very rapid recurrence (within one year) in this case would seem to substantiate the impression.

Previous Lactation. Twenty-two patients, or a little over 30 per cent, of those where mention is specifically made regarding this point, never lactated. It would seem as though there were some predisposition to carcinoma in breasts that have not lactated.

Duration of the Tumor Prior to Treatment. The average period elapsing between the time the patient first noticed the tumor and her entrance into the hospital for treatment, was six months. It is interesting to note that in spite of propaganda and the employment of every means known to educate the laity and physician regarding the significance of a "lump" in the breast, the duration of the tumor mass in patients admitted in 1915 averaged six and a half months, whereas in 1924 the duration was six months, hardly sufficient difference to make one feel justified in telling that any real progress has been made along these lines. It is interesting to note that, of 5 patients followed at least five years, a history of a tumor mass existed in 1 case two and a half years; in 2 cases five years and in 2 cases eight years; 1 patient lived nine years and then died of a recurrence, 2 are alive and free of visible recurrences at the present writing, and, finally 2 promptly developed recurrences. The longest duration of a tumor mass, twelve years, occurred in a male patient without glandular involvement. This patient died three years after a radical operation of a recurrence. Broders first called our attention to what he calls the "index of malignancy," based upon cellular differentiation. Sistrunk and MacCarty, in an analysis of a series of breast carcinomata based upon a histologic study of the neoplasm, arrived at the conclusion

that the duration of life following the operation was in no small measure dependent upon cellular differentiation, hyalinization and fibrosis. It would certainly seem as though some factors inherent in the neoplasm, or perhaps in the patient herself, predetermined some of these findings.

Location of the Tumor. In 116 cases, the tumor occupied the left breast; in 102 the right. The location of the growth designated according to breast quadrants was as follows: 90 occupied the upper and outer quadrant; 22 the upper and inner quadrant; 24 the nipple region; 10 the lower and inner quadrant and 12 the lower and outer quadrant. In the remaining cases no definite mention of the breast quadrant harboring the tumor was made. The relationship of the position of the tumor to lymphadenopathy will be discussed in considering lymph-node involvement.

Pathology. The various tumors were all examined by the pathologist of the hospital, Dr. F. S. Mandlebaum, and were classified as is shown in Table III, which also shows the attendant lymphadenopathy for the entire series.

TABLE III

Type of carcinoma	Number of cases.	Lymph nodes involved.	Lymph nodes not involved.	Not mentioned.
Scirrhus	101	67	29	5
Medullary	43	26	15	2
Medullary and scirrhus	15	11	3	1
Adeno	14	6	3	5
Duct	14	9	5	0
Duct and papillary	10	2	6	2
Carcinoma (unclassified)	6	2	3	1
Papillary	5	0	4	1
Papillary (from ducts)	3	0	3	0
Papillary cystadeno	3	0	3	0
Gelatinous	1	1	0	0
Colloid	1	0	1	0
Carcino-sarcoma	1	0	0	1
Spheroidal	1	0	1	0
	218	124 (56%)	76 (35%)	18 (9%)

Table IV shows the type of tumor in the 89 cases followed at least five years, as well as the percentage of those alive and well, January 1, 1926.

TABLE IV

Type of tumor.	Number of cases.	Percentage.	Number of patients alive and well, followed at least five years.	Percentage.
Scirrhus ca.	42	60	8	19
Medullary and scirrhus ca.	7	10	1	14
Medullary ca.	8	11	3	37
Duct ca.	4	6	1	25
Papillary ca.	4	6	1	25
Adeno-ca.	2	3	1	50
Carcino-sarcoma	1	1+	0	
Papillary cystadeno-ca.	1	1+	0	
Gelatinous ca.	1	1+	0	
	70*	99		

* The discrepancy in pathologic reports and number of patients followed (89) is accounted for by the fact that some were classified merely as carcinoma, and in a number of the recurrent cases the pathologic report is missing.

Table III reveals the different pathological types of breast cancer met with in this series, as well as the lymph-glandular involvement for each type, as encountered at the operation. Table IV shows the different pathologic types for 70 of the 89 patients followed for at least five years, with the percentage of each type and the percentage of patients alive and well in each group. Many of these groups are much too small to permit conclusions as to the relative benignity or malignity of one type of carcinoma as opposed to the other. There is one group, however, commonly classified with the breast carcinomata which seems to be relatively nonmalignant. We refer to the papillary cyst adenocarcinomata. In this series we encountered 3 cases: 2 of the cases were treated by simple mastectomy and will be referred to later; 1 died of a recurrence six years after a radical amputation of the breast.

Glandular Involvement. Reference to Table III shows that 56 per cent of the entire series revealed lymph-node involvement; 35 per cent showed no evidence of lymph-node involvement; in 9 per cent the presence or absence of lymph node was not mentioned. Of 139 cases, followed for a period of from two to eleven years, 59 per cent showed lymph-node involvement; 30 per cent were uninvolved and in 11 per cent no mention was made of the presence or absence of involvement. Of 218 patients, 106 gave palpatory evidence of some lymphadenopathy; the

pathologic findings, however, did not substantiate this in all the cases. It seems as though the tumors situated in the upper and outer quadrants of the breast gave a relatively higher proportion of glandular involvement, but our findings here are not in sufficient numbers to warrant a definite statement to this effect.

Follow-up Results. We have attempted to simplify the interpretation of our follow-up results by combining in one chart (Chart 1) the number of patients we were able to follow each year, beginning with the year 1915 and continuing through 1924. At the top of the chart we have in sequence the number of years it was possible to follow each group. We have, moreover, subdivided each year so that deaths and recurrences could be tabulated in the order in which they occur. These, subtracted from the original number, or from the number of patients alive and well at the end of any given yearly period, immediately reveal the number of patients still alive and well. Summarized, our effort at follow-up shows the following:

Of 139 cases followed one year, there were 21 deaths, 5 recurrences, 113, or 81 per cent, living.

Of 139 cases followed two years, there were 23 deaths, 3 recurrences, 86, or 62 per cent, living.

Of 125 cases followed three years, there were 12 deaths, 1 recurrence, 66, or 52 per cent, living.

Of 115 cases followed four years, there were 14 deaths, 2 recurrences, 48, or 41 per cent, living.

Of 89 cases followed five years, there were 5 deaths, 0 recurrences, 31, or 34 per cent, living.

Of 73 cases followed six years, there were 6 deaths, 0 recurrences, 23, or 31 per cent, living.

Of 56 cases followed seven years, there were 1 death, 1 recurrence 15, or 26 per cent, living.

Of 37 patients followed eight years, there were 2 deaths, 1 recurrence, 6, or 16 per cent, living.

Of 31 patients followed nine years, there were 2 deaths, 0 recurrences, 2, or 7 per cent, living.

Of 26 cases followed ten years, there was 1 death, no recurrence, 1, or 4 per cent, living.

Of 14 cases, followed eleven years, there was 1 death (intercurrent); 0 recurrence, 0, or 0 per cent living.

It will be noted that at the end of a five-year follow-up 31

patients were alive and well out of a total of 89 cases followed. If we were to stop here and consider these patients as cures, our results would compare favorably with the results of other authors (Table V).

TABLE V

Author.	Number of cases.	Percentage alive after five years.
Judd and Sistrunk	514	38.0
Porter	22	27.7
Sistrunk	218	36.7
Bunts	341	26.9
Greenough and Simmons	69	32.0
Primrose	76	44.4
Deaver	150	26.0
Moschcowitz	89	34.0

I must confess I had always thought, in spite of numerous reports of late recurrences after operation, that a case of breast carcinoma alive and well five years after operation could almost be classified as a cure. I find, however, on the contrary, that in the sixth, seventh, eighth, ninth and even tenth year after operation, recurrences; to be sure, most of them metastases, which have claimed no less than 16 patients out of a total of 31 who at the end of a five-year follow-up period were considered well. In 1 of these death was due to an intercurrent disease; 1 of these patients is alive with a recurrence. In all the others death was due directly or indirectly to carcinoma. *Of* 89 *patients followed at least five years and in some cases for a much longer period,* 15 *at the present writing are alive and well, or a percentage of* 17.

Influence of Glandular Involvement on the Prognosis. Of 53 cases dead of carcinoma or with a recurrence within three years after operation, 81 per cent had pathologically involved glands; 19 per cent did not. Of the cases living four to five years, 60 per cent showed no glandular involvement, whereas 40 per cent did. Of cases living six to eight years there were an equal number with and without glandular metastases; whereas of those few patients who lived, nine, ten and eleven years, all had glandular metastases. We were surprised to find that of those patients who lived for a period beyond five years, 50 per cent had axillary glandular metastases, for we were led to believe that this was most unusual. Whether this is due to the small series here presented or to the possible limited extent of the axillary involvement, is conjectural.

Recurrences and Metastases. It is not an easy task in a series of this kind—where reports of deaths are obtained either from relatives, social workers or from sister institutions who care for incurables—to establish with accuracy the number of local recurrences as opposed to distant metastases. The data, however, at our command showed the following: In the first year, 5 patients developed local recurrences; in the second year, 4, and in the succeeding years there was a corresponding decrease in the number of local recurrences in contradistinction to the number of deaths from distant metastases. For example, of those patients followed at least six years, 6 died of carcinoma during that year, but none of these, as far as our records show, developed local recurrences. The following distant metastases occurred in this series. Lung and pleura, 6 times; spine, 3 times; supraclavicular nodes, 3 times; liver, 2 times; peritoneum, 4 times. This must give rise to speculation and leads me to repeat what, I believe, I have expressed before this Association on a previous occasion, namely, that those patients who develop distant metases, in the absence of any visible evidence of local recurrence, probably had extensions of the process into lymphatics beyond the operative field before they submitted themselves for treatment. In other words, the surgeon, in spite of the most radical operations, faces a condition against which he can hardly hope to cope. On the other hand, the question of local recurrence, I believe to be intimately bound up with the conditions as found at operation and these will vary in the main with the degree of axillary lymph glandular involvement, adherence of glandular masses to the axillary vein or to the skin of the axilla or any other factor or factors which makes the surgeon feel, for one reason or another, that conditions were not suitable for a really radical operation. It is interesting to note that in the few cases in which the prognosis as to life expectancy or recurrence was indicated by the surgeon at the time of the operation, his judgment was borne out by subsequent events. We have not found that supraclavicular fulness following the radical operation is necessarily indicative of a beginning or established supraclavicular involvement, but rather believe that it is, in most cases, the result of a lymphedema which involves the skin and subcutaneous tissues of the supraclavicular triangle. We have followed over a period of years a number of patients who have marked supraclavicular fulness without any palpatory evidence of

a recurrence here. On the other hand, its presence should make one suspicious of the possibility of supraclavicular lymph-node involvement.

Ulcerating and Inflammatory Carcinomata. We have encountered three cases which answer the description of inflammatory carcinoma, as described by Lee and Tannenbaum. All these patients developed prompt recurrence and a generalized carcinosis within six months after operation.

Bilateral Mammary Carcinoma. Two cases presented themselves with what we took to be consecutive bilateral carcinomata. One patient is alive and well fourteen years after the first mastectomy and seven years after the second. The other patient developed her second carcinoma three and a half years after her first operation and died of a recurrence four years after the second operation.

Serohemorrhagic or Hemorrhagic Discharge from the Nipple. Five patients of this series presented themselves with a bloody discharge from the nipple. In each instance a duct papillary carcinoma, or a papillary cystadenocarcinoma, was reported. In a similar length of time the records show that 9 patients admitted with a serohemorrhagic discharge possessed benign duct papillomata. One of us proposes to review this series.

Simple Breast Amputation. In three instances (these are not included in the series) a simple mastectomy, or local excision, for a papillary cystadenocarcinoma of the breast was done. One patient followed for two years was alive and well, when she was lost sight of. One patient is alive and well four years after operation; the third patient could not be traced.

Preoperative and Postoperative X-ray Therapy. In six patients of those we have been able to follow, prophylactic *x*-ray radiation was employed prior to the operation. Three of those are alive and well for periods of two, three and five years. Three have had recurrences. It is almost impossible for us to accurately evaluate the benefits of either preoperative or postoperative radiation. We are employing postoperative radiation almost as a routine measure at the present moment, but have seen recurrences begin and increase in size in areas treated most intensively. Too many other factors enter into consideration. A number of patients who are now alive and well, and who are included in the

follow-up statistics, have received the benefit of postoperative radiation. How much this has influenced longevity is conjectural.

CONCLUSIONS. 1. A follow-up of three years after amputation of the breast cannot be considered as of great value.

2. Even a follow-up of five years cannot be considered as absolutely conclusive; too many patients die after this period from recurrences or metastases.

3. For all practical purposes the fate of the patient is sealed at the time of the operation. The prognosis depends upon three factors: (*a*) The local extent of the process; (*b*) the presence of distant metastases; (*c*) the care with which the operation is done.

4. The modern operation is usually successful in eradicating the local process, as is evidenced by the very large number of cases dying from distant metastases without even a suspicion of a recurrence.

5. At the present writing we are practically powerless in the presence of distant metastases.

6. The late results of the operation are not as favorable as one might be led to believe from a cursory examination of the literature.

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DISCUSSION

Dr. Robert B. Greenough, Boston, Mass.:

The deductions to be drawn from Dr. Moschcowitz's table are certainly not encouraging. My own experience has lead me to believe that the end-results of operative treatment were better than those he has given. We have a chart on which we have arranged in graphic manner the rate at which three different series of cases of cancer of the breast have died, representing the lapse of years from the time of discovery of the tumor on the lower horizontal line and the number of cases on the upright, arranged on a percentage basis. All three series start with 100 per cent living at the time of the discovery of the tumor.

The first series comprises 16 early and favorable cases of cancer of the breast in which the axillary glands were not involved. All of these cases were treated by radical operation and 67 per cent were living at the end of seven years from the time of the onset of the tumor.

The next series included 66 cases of cancer of the breast, both favorable and unfavorable, that is, with and without involvement of the axillary glands, and of these cases 37 per cent were living and well seven years after the onset of the tumor. About one-half of these cases received postoperative prophylactic *x*-ray treatment, but with no better results than in the cases which did not have *x*-ray.

The third series was collected by Dr. E. M. Daland, from the records of the Huntington Hospital and the House of the Good Samaritan. It includes 100 untreated cases of cancer of the breast. This series gives us a point of departure with which to compare the results of treatment. Five years from the onset of the tumor 25 per cent of these untreated cases were living and even at the end of ten years the percentage had gone only as low as four.

It is from this table that I have deduced the fact that surgery was of material benefit in the successful treatment of cancer of the breast.

Dr. Frank E. Bunts, Cleveland, Ohio:

I am very much interested in this paper by Dr. Moschcowitz, especially in the chart which he showed us, which I think is a very clear way of determining the relative longevity. It seems to me we are really missing the true state of affairs when we try to estimate longevity by the late results of a group of cancers taken indiscriminately without reference to their relative malignancy.

We know very well that cancers have different grades of malignancy, and when we group some of these soft encephaloid growths together with the small, hard scirrhous, I believe we are doing an injustice to our statistics; at least we are confusing them.

If we could divide cancers into four groups as the Mayo Clinic does, and follow the statistics of these four groups, I think we would arrive at quite a different estimate of the longevity after different types of operations. It is quite conceivable that one operator might have a predominance of cancers of one kind, while another operator would have more of another kind. While the statistics of different operators are approximately the same as to five-year nonrecurrences, yet if their cases were grouped according to malignancy I think we would find very different figures. Certainly five years is a very short period when we consider nonrecurrence for this period as a basis of possible cure. Everyone knows there are late recurrences. While after that period I think the recurrences are chiefly distant metastases, yet, of course, we have a certain number of local recurrences.

Yesterday a doctor reported a case in which numerous operations had been performed, and finally a plastic operation upon the chest to cover a defect in the chest wall. This was a type of case, as Dr. Mayo suggested, in which probably the malignancy was relatively slight and in which future operations would promise considerable relief.

Metastases occurring after ten or fifteen years are not very infrequent, and while one might think that when they occur in the bones, the spine particularly, they represent a different growth, we can hardly consider them as anything else but as metastases from the original growth.

I have heard a good deal said about operating upon cases of cancer in which there was no axillary involvement. I confess that in my own experience it must be that I have received bad cases, because almost without exception I have found axillary involvement. While the axillary glands may not be palpable at the time of the operation, the microscope reveals their involvement. It is not wise, I think, to make the statement, as some have done, particularly my friend Dr. Lee, that those cases in which there is no axillary involvement are those that are perhaps best suitable for *x*-ray or radium treatment, because the axillary glands are nearly always involved in the type of cancers that come to our clinic.

In regard to the operation itself, I hesitate somewhat to make this statement, but I have been convinced by the experience of Crile, my associates and myself, that the extremely radical operations are not necessary, and I have scarcely ever found it necessary to remove so much of the integument that it could not be closed by sutures or clips. Indeed, in our subsequent treatment of local recurrences our *x*-ray department has assured us that most of the local recurrences in the form of little nodes which are found after these operations nearly always occur at the point of the stitch punctures, and so I try now to leave enough skin so that I can bring it together and use not more than one or two silkworm sutures.

In cancers situated in the upper outer portion of the breast I can scarcely conceive of the value of the extensive operation suggested by Handley, viz., going down and dissecting out the sheath of the rectus. If the cancer has extended that far from the upper outer portion, and if the cancer extends by permeation through the lymphatics and in a radial direction, then it must be that the cancer has extended far beyond any region which we could hope to reach by any operative procedure.

So far as *x*-ray is concerned, we have tried the deep *x*-ray with high voltage, preoperatively, and we have found that the cancer returns much more rapidly in those cases than it does where it is not used. So this form of treatment has been abandoned. Whether it should be used postoperatively or not I think is a moot question.

In going over the records, I find that we have an almost equal number of five-year "follow-ups" among those cases which have received *x*-ray postoperatively and among those which have not, and that there is only a fractional percentage difference between the five-year cases of nonrecurrence in these groups.

Dr. William L. Estes, South Bethlehem, Pa.:

It occurred to me that in our study of the after-results from operations on carcinomata, especially of the breast, we have neglected one very important investigation, and that is to try to determine whether the fact of a former infection by carcinoma, especially of the breast, has reduced the general resistance of the patient to intercurrent disease.

Some time ago the Mayo Clinic made a very interesting study of the results, as shown by insurance data, in regard to the expectation of life from patients who have been operated upon for cancers of the duodenum and for ulcers of the duodenum and stomach. They found the expectation of life was rather increased.

Is it a fact that the expectation of life, or the expectation of the intercurrent disease taking off the individual after carcinoma of the breast, is reduced very much? I do not think anybody has made a thorough study of that.

We all have cases of long life after operation for carcinoma. In reviewing some of my cases I found one who has lived seventeen years and is free of any recurrence. There was another in which, after recurrence, it was necessary to remove a large area of the anterior wall of the chest, and which, by the way, I plugged by using the lung, simply catching up the lung and sewing it to the inner surface of the chest wall, afterward grafting skin over it. She made a perfectly good recovery and lived for fifteen years. Remember, that is a recurrent case. Were those two women especially resistant? Had their expectation of life, contrary to the rule, not been reduced? And if it is the rule, can we by some process of study or investigation find out whether

the expectation of life after carcinoma of the breast is very much reduced aside from the recurrence?

We all have a very large number of cases, in the aggregate, which have gone over five years. After that five years we say that the resistance to cancer has been established, she will probably not have a recurrence. But will she be taken off by some other disease quicker than another individual who never had carcinoma?

Dr. John L. Yates, Milwaukee, Wis:

Biologic concepts of cancer may no longer be disregarded. They show that cancer has basic characteristics common to the other chronic lymphomatous diseases and that comparative pathology will provide means the better to understand and to combat cancer. These chronic parasitic diseases, and in carcinoma the cancer cell is the parasite, are contests within living hosts between offense, the focal and disseminated irritants emanating from parasites, and defense, the local and systemic responses to the irritations. All who escape untimely death from other causes are subject to infection with parasites or to the irritations which change relatively normal epithelial cells into cancer cells. Prophylaxis, therefore, can never be entirely effective. Treatment consists of three steps: Reduction in offense, *e. g.*, radical extirpation of cancer, augmentation of defense and conservation of patients' energies, so as to enable them to withstand the stresses of radical treatment and to maintain subsequent health.

The discussion at this session has been confined to what can be done *to the cancer.* What may be done *for the patient* has received no consideration. The reason is definite. We have accepted the dogmas of those students of cancer who, because they do not study patients suffering from cancer, deny the existence of a defense against cancer and would have us believe the impossible, namely, that in Nature there arises an action without a reaction, an offense without a defense.

Facts refute this doctrine. Local irritations are proved by the inflammatory responses which include cellular infiltration and proliferation of fixed tissue cells which form the fibrous tissue that is subject to subsequent degeneration. The efficacy of defense is manifest in the types of proliferation of the atypical epithelial cells. The less the defense capacity, the more the cells deviate from normal and the more bizarre the tumor. More recently MacCarty and his co-workers at Rochester have found that these responses are reliable indices of the life expectancy of patients after operation.

Systemic irritations provoke the alterations in the numbers and proportions of blood cells shown by blood counts and in the constituents of the plasma exhibited in hastened rates of sedimentation. Both of these alterations tend to disappear as patients approach recovery. No

more incontestable evidence of systemic irritation can be found than the cachexia which develops as both cells and plasma are deteriorating. It were well to recall that Peyrilhe, in 1776, recognized that cachexia was the result of noxious substances produced by the neoplasm. Further, the contest between offense and defense fluctuates as it does in other maladies. Witness the periodic variations in temperature, in the sizes of tumors and in the three fairly well-defined stages which are directly comparable to those pictured by Trousseau in his classic description of the malignant lymphomas.

Students of cancer have refused to see the import of the manifestations of the natural processes antagonistic to the progress of cancer and have failed to utilize them to help patients. Dr. Greenough's diagrams serve to illustrate what has been accomplished, to emphasize failures and to indicate means to improve the benefits conferred.

The first curve indicates the death-rate in 100 patients suffering from cancer of the breast when the progress of their disease was unmodified by treatment. Those of least resistance died earlier. Others were possessed of such robust defense that one-fifth were alive after seven years and one-tenth survived a decade. These patients had types of disease that would be termed favorable and unfavorable. All who escaped death from other causes were doomed to be destroyed by cancer.

The second curve shows the death-rate in 66 patients subjected to operations that at least were called radical. The disease was unfavorable in type or extent. The curve parallels the first curve until the fifth year, when the descent is less rapid, so that at the seventh year one-third are apparently well. Thus merely subtracting from offense benefits materially those patients of more limited life expectancy but whose defense capacities were less impaired. Dr. Moschcowitz's evidence justifies an assumption that all patients in this group will die of cancer unless otherwise destroyed.

The third curve is based upon the death-rate of 16 patients subjected to supposedly radical operations who had types and extent of cancer that constituted less unfavorable prognosis. At the end of seven years two-thirds had remained well as compared to one-fifth in the first series and one-third in the second. A proportion of these survivors will die of cancer, some as late as thirty years after operation. Another proportion have achieved permanent recovery; they have attained the status of insuceptibility.

What can be deduced that is of direct practical value for those who would further the effectiveness of cancer therapy?

The minority of individuals are susceptible to cancer because they are deficient in resistance, defense and repair. Degrees of susceptibility and rapidity of subsequent progress of the disease are commensurate with the extent of that deficiency.

Two phases are noteworthy in the pathogenesis of cancer—the formation of the initial lesion (structural cancer) and the dissemination of cancer cells from that lesion (biologic cancer). They may be almost synchronous or a decade may separate them.

Cancer cells, like tubercle bacilli, are perpetually and widely disseminated. Many are destroyed; some survive, often inactivated, but retain their pathogenic capacity, so that years after they can reproduce and, in cancer, form metastases. Both diseases are incurable but recoveries from both can be promoted.

The first step in treating cancer is to eradicate the initial lesion together with all regional glands because they are more likely than not to contain cancer cells, even though undemonstrable. This Halsted proved would lead to permanent local freedom from disease, and, as Dr. Moschcowitz has again emphasized, local freedom is the established requirement for lasting benefit.

The second step, which Dr. Moschcowitz laments is not available, is to arouse and to maintain the body defense needed to continue the inactivation of cancer cells already disseminated as emboli, so that they do not multiply and form metastases.

We may turn to biology and to the world's experiences of tuberculosis for methods. Local lesions, the primary and secondary tumors, are the battlefields whereon the campaigns against disease are won or lost. The outcome of the battles is determined almost exclusively by the reinforcements brought to the fixed tissues, in other words, by the quality and quantity of blood delivered to them.

The divers cells concerned in the production of blood corpuscles and plasma receive their energies from two sources, ingesta and sunshine. They are subject to fatigue from overwork and to incapacity from intoxication.

Hence, if defense is to be raised and conserved, rest, proper food and sunshine are essential. In short, the régime developed by tuberculosis therapeutists, including Rollier, which imposes the burdens of hygienic living upon patients, should be followed. These are the simple means most needed to supplement operation, which begins treatment that should be continued henceforth. We believe we have observed benefits thus attained, benefits that cannot otherwise be provided.

The greatest obstacle to progress in treating cancer is the word "cure." Once it is recognized that cancer is incurable, and that efforts are to be concentrated upon how patients may be provided with opportunities to attain recovery, then much will have been gained. We will cease to delude ourselves with the immodest belief that we possess divine attributes which permit us to cure the incurable. We will gain in self-respect as we stop a pose that merges with that of charlatans. We will gain the respect and confidence of our patients who will profit greatly.

Dr. W. E. Sistrunk, Rochester, Minn.:

I have had an opportunity to study two large groups of patients operated on for cancer of the breast, and I feel Dr. Moschcowitz is right in stating that we should not infer from the statistics which we publish that the patients mentioned as being alive are necessarily cured; they should be looked upon as patients whose lives had been prolonged up to the time that we were able to prove that they were still alive.

I believe that we shall never, through surgical methods, be able to improve very much the results which we now obtain in treating cancer of the breast. The operation which was devised a number of years ago by Halsted and Meyer has never been improved on to any extent, and I think our only chance of improving the end-results in cases of cancer of the breast will be through operating earlier.

The prognosis in an individual case depends largely on the type of growth and the involvement or noninvolvement of the lymph nodes at the time of the operation. There is no doubt that we see certain scirrhous types of carcinoma which grow and metastasize very slowly. On the other hand, we see the medullary types of growth in which all effort toward differentiation on the part of the carcinoma cells has been lost, and these are certainly very highly malignant; as a rule, patients with such growths cannot be expected to live. The reaction of the tissues in the neighborhood of the growth seems also to have a bearing on the prognosis. Some years ago MacCarty and I published a paper concerning these reactions and were able to show that better results followed operation in cases in which a large amount of hyalin tissue and an extensive fibrosis were present in the tissues surrounding the growth.

From my own experience, when the lymph nodes are found involved at operation the chance of permanent cure is much diminished. In some such cases life is probably prolonged by the operation, but when lymph nodes are found to be involved I believe that liquefaction must already have taken place in the growth; when this has happened any or all of the lymphatics which drain the breast are likely to contain this liquid cancerous material.

The lymphatic vessels supplying the breast usually accompany the bloodvessels to the breast, and when evidences of involvement of the axillary lymph nodes are found the chances are much in favor of the disease having already spread to other lymphatics, such as those accompanying the intercostal vessels, the internal mammary artery and so forth.

It is impossible always to determine clinically whether distant metastasis has occurred. I operated recently on a patient with extensive carcinoma of long duration; the axillary lymph nodes were involved, but it was still possible to perform a radical operation. Pulmonary

embolism, about ten or twelve days after the operation, resulted in death. A careful examination had been made by roentgen-ray, and so forth, before the operation to determine whether internal metastasis had occurred; the results were negative, but necropsy revealed extensive involvement of the lymph nodes in the mediastinum.

One interesting finding was noted in the last group of cases that I reported. In 60.5 per cent of these patients who returned with local recurrence internal metastasis was found. This would indicate, I think, that when the lesion recurs locally it has usually recurred internally; nearly all such patients eventually die from the disease.

In working up statistical data we find that when we continue to write to patients operated on, to their doctors and to their relatives, we are able to show a greater and greater number alive. Changes in patients' addresses prevent us from communicating with many who are still alive, and hence we cannot include such patients in our statistics as being alive.

Dr. Miles Fuller Porter, Ft. Wayne, Ind.:

Personally I have been having forced slowly, and I confess somewhat reluctantly, upon me the idea that an individual suffering from cancer, we will say, of the breast is not only suffering from a tumor or a lump situated in the breast, but, of more importance still, is suffering from a something—call it lack of resistance, if you please—behind that lump which makes it possible for that lump to develop. With that feeling, it seems to me our greatest hope for further improvement in the treatment of these malignant tumors lies not in changes in technic or perhaps not even so much in time of operation as in finding, if we can, how to improve this resistance that is spoken of, and perhaps by and by start to improve it by breeding it out.

There is one other point that I should like to make. I should like to refer to the dangers of *x*-ray treatment in cancer, preoperative, postoperative or to the exclusion of operation.

These dangers are of three kinds: (1) The production of a local malignant growth from apparently the direct action of the *x*-rays; (2) the systemic effect that results from intoxication; and (3) the condition of fibrosis which is produced by the so-called intensive therapy.

I have had two patients killed with intensive *x*-ray therapy. One of them died a slow death by a process of fibrosis which reduced her to the point where she had a small portion only of one lung left. The other one died as the result of what might be called an acute toxemia as the result of one application of *x*-ray treatment.

Dr. Moschcowitz (in closing):

Primarily permit me to express my thanks for the very liberal discussion which was accorded to my paper, on so trite a subject as car-

cinoma of the breast is. Owing to the brevity of the time allotted to me, I could not quite cover the ground. I did it so hastily that I doubt whether I made myself clear. I would in consequence like to emphasize the following:

I am heartily in favor of the propaganda of the Association for the control of cancer. How futile these efforts are, however, is shown by the fact that prior to these organized efforts the average duration of the lump in the breast before the patient came under our observation was six and a half months, while since that time the period was reduced to six months; a reduction of half a month, or a period hardly worth while noting.

I would also like to emphasize all that Dr. Porter has said regarding *x*-ray therapy. I personally have abandoned long ago all preoperative radiation. I believe the results are bad enough if the patient is operated upon immediately, and see no reason for delaying the operation for three weeks unless it is definitely proven that the results are so much superior with preoperative radiation, and they have not been so in my hands. I salve my conscience by permitting postoperative radiation, although I confess I very much doubt its efficacy. As I have stated on a previous occasion, I am exceedingly conservative in everything that pertains to surgery. I am, however, exceedingly radical both in my views and operations for carcinoma. If we were asked the question, what can we do for an individual afflicted with a carcinoma of the breast, we would have to reply that even with a very radical extirpation we can at best remove the carcinoma locally. This is proven by the fact that many of these patients are alive and well without *x*-ray therapy. In other words, they do not die of local recurrences; as a matter of fact I have found that local recurrences are rare after the modern operation for carcinoma of the breast. If the patient dies she usually dies with and from distant metastasis. In the presence of this, however, both with and without radiotherapy we are, according to my opinion, almost powerless.

SYMPATHECTOMY IN RAYNAUD'S DISEASE, ERYTHROMELALGIA AND OTHER VASCULAR DISEASES OF THE EXTREMITIES

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Surgery is now in the midst of a period of enthusiasm over operative procedures directed toward the sympathetic nervous system. A voluminous literature deals with the practical results of periarterial sympathectomy in a wide variety of diseases, but particular those in vascular and trophic lesions of the extremities. Because of the paucity of our knowledge of the anatomy and physiology of the sympathetic nervous system, there is necessarily a mass of empirical clinical data. It follows that our knowledge of the rôle of the sympathetic system in pathologic conditions is likewise meager. Our attention was first directed to the peripheral effects of sympathectomy in an experimental and clinical study of the results produced in spastic diseases of the extremities by removal of the sympathetic trunks, their rami and ganglia. We wish to record our experiences and impressions obtained in removing the influence of the sympathetic innervation to an extremity in Raynaud's disease, erythromelalgia and other vascular diseases of the extremities.

Anatomy and Physiology. It would seem fundamental to have clearly in mind in just what manner the bloodvessels of the extremities receive their innervation. The physiologic work of the nineteenth century brought out the now commonly

accepted fact that the muscular action of the bloodvessel walls is under the control of the sympathetic nervous system.

It will be remembered that the sympathetic nervous system is composed of a craniosacral and a thoracicolumbar outflow. These two portions differ from one another anatomically, physiologically and in their reactions to the administration of drugs. The craniosacral outflow has received the name "parasympathetic" in contradistinction to the thoracicolumbar division which has been termed the "sympathetic" division. We are concerned with the thoracicolumbar outflow of fibers. Within the intermediolateral cell column of the anterior horn of gray matter in the thoracic and lumbar portions of the spinal cord are found the cells of origin of the fibers of this division of the sympathetic nervous system. These fibers leave the spinal cord through the anterior or motor roots and reach the sympathetic ganglia situated paravertebrally. Here they end to form a synapsis with the dendrites of a sympathetic ganglion cell. These small, myelinated fibers constitute the preganglionic sympathetic nerve fibers and make up the white rami communicantes. From the sympathetic ganglion fibers arise which join the spinal nerves. These postganglionic fibers are unmyelinated and form the gray rami communicantes. Some of these postganglionic fibers proceed directly to the structures which they innervate, while the majority join the spinal nerves.

It will be seen that as far as our knowledge now goes the sympathetic nervous system is purely an efferent one. The possibility of antidromic *afferent* conduction will be spoken of later.

The vessels of the extremities receive a sympathetic innervation through the somatic nerves and this supply is received at regular segmental levels. In addition there may be a direct supply from the sympathetic ganglion cells by a fine plexus of fibers within the sheath of the arteries. The question of the existence and importance of these two types of innervation has been a mooted one for many years. Kramer and Todd[15] studied the distribution of the vasomotor nerves to the arm and Potts[22] made a similar study upon the leg. In both instances the conclusions were identical; that the nerve fibers are supplied to the bloodvessels at regular levels in their course and that only a small number of fibers accompany the large arteries to end in the arterioles and capillaries. Langley[17] stimulated the lumbar sympathetic chain before and after section

of the femoral and sciatic nerves. Pallor of the foot was obtained before cutting these nerves and was abolished after section. He also injected ergotoxine and then stimulated the lumbar sympathetic chain. Upon the side of the cut peripheral nerves there was no effect, while flushing occurred upon the opposite side. He concluded that the vascular sympathetic fibers run to the trunk and limbs by the somatic spinal nerves and not by way of the arteries.

Milko[20] stimulated the sheath of the femoral artery in dogs and secured no changes in volume of the leg. However, vasodilatation was obtained after the sciatic nerve was severed. Schilf[24] secured no increase in the cannulated bloodflow in dogs upon stimulation of the sheath of the femoral artery. Bowing[2] made plethysmographic studies upon the dog's leg and found that all of the vasomotor reflexes are maintained after periarterial sympathectomy and are abolished by interrupting the mixed peripheral nerves. He stated that the fibers for these vascular reflexes do not accompany the vessels, but join them only upon reaching the periphery. Wiedkopf[27] has shown that anesthesia of the brachial plexus in man produces the same changes in the arm as does removal of the sympathetic chain. These effects cannot be obtained by removing the adventitia of the vessels. Denning[8] sectioned the femoral nerve below Poupart's ligament and the sciatic nerve in the middle of the thigh in dogs. He then exposed the femoral artery and vein and drew a rubber tube beneath them. He ligated the extremity *en masse* without including the vessels. After these procedures he injected barium chloride into the tibial artery. Contrary to the normal result, there was no painful reaction. He concluded that the sensory nerves of vessels of the lower extremity run with the spinal nerves. Joris[14] has insisted upon the presence of a perivascular nerve plexus which anastomoses along the entire length of the vessel. He described the presence of sympathetic ganglion cells within this perivascular plexus. Diaz[9] states that these are not true ganglion cells, but are entirely different from sympathetic ganglion cells in their structure and size. He states that they are similar to those cells found in the sinusoidal node and in the wall of the auricle of the heart. Guillaume[11] has carried out very careful microscopic disections of the larger vessels of the extremities. He believes that stripping the wall of an artery does not completely remove the innervation to that

vessel. He states that removing this adventitia over a small length of the vessel cannot possibly interrupt fibers going to the rest of the extremity. This author admits that there is a fine plexus of fibers in the sheath of the peripheral arteries, which divides into innumerable branches, but strongly emphasizes that the majority of the vascular innervation comes to the vessels in a segmental distribution from the spinal nerves. In this connection it is interesting to note that in median nerve lesions Crile[7] has recently called attention to the development of cyanosis, accurately delimited to the cutaneous area supplied by the median nerve, following the application of the cuff of a sphygmomanometer to the arm and inflation to equal the patient's diastolic blood-pressure. This phenomenon disappeared as evidences of regeneration of the nerve appeared.

From a study of the literature on the subject it would appear then that there can be no question that the peripheral vessels receive their major sympathetic innervation by a segmental distribution from the spinal nerves and that these fibers originate from the sympathetic ganglia of the thoracicolumbar division as postganglionic fibers. There is a serious doubt as to the presence of a sympathetic innervation in the adventitia of the vessel which continues throughout its course. It would be difficult to conceive of how under such circumstances the removal of a small portion of the vessel adventitia could affect the caliber of the vessel at a distance. However, we must take into consideration the reports of hyperemia and temperature changes produced in the hands and feet of patients after such an arterial decortication.

In 1913, Leriche[19] called attention to the results he had obtained in man by thoroughly removing the periarterial sympathetic fibers about the femoral artery. He called particular attention to a hyperemia produced by vasodilatation and to an increased warmth of the extremity undoubtedly produced by the same mechanism. Leriche reported the beneficial and curative effects of such a procedure in many clinical entities. He was quite unable, however, to verify these findings upon experimental animals. Nevertheless, Leriche has stated that in man the predominating vasomotor influences are carried by the perivascular sympathetic nerves.

In considering the clinical results obtained by supposedly interrupting the sympathetic supply to the bloodvessels, one is immedi-

ately struck by this complete absence of any basic experimental physiologic work upon which the beneficial results may be explained. Certainly nowhere is there any experimental confirmation of Leriche's statement that in man the predominant vasomotor impulses are carried in sympathetic fibers found in the bloodvessel adventitia.

Callander[4] found no rise in the systolic blood-pressure in man after arterial decortication and in only one case could he find an increase in temperature in the operated upon extremity. Lehman,[18] working upon animals, could not corroborate the findings of vasodilatation, increase in blood-pressure or temperature in the limb operated upon. He also denied any beneficial effect of arterial sympathectomy upon wound healing. Bowing's and Wiedkopf's[2,27] plethysmographic studies upon animals failed to confirm any of the reported results following periarterial sympathectomy. Elving[10] directly examined the capillaries following decortication of the vessel walls. He noted improvement of the circulation in 4 cases; a decrease in 2, and no change in 6. Any of these results may be explained by Sherrington's work, in which he demonstrated that freshly excised arteries show different diameters under the same wall tension when they have no sympathetic connections. Mosser and Taylor[21] performed an arterial decortication upon 10 cats and 5 dogs. In only 2 did they find an appreciable temperature increase.

This experimental work merely confirmed Langley's statement that all vasomotor impulses leave the spinal cord by way of the white rami, reach the sympathetic ganglia, are relayed through the gray rami to the spinal nerves, and thus reach their peripheral destination. It came to be believed that dilatation of arteries is produced only by paralysis of the normal vasoconstrictor fibers which have their origin in the sympathetic ganglion cells.

In 1874 Goltz sectioned the sciatic nerve and obtained a vasodilatation and a rise in temperature in the corresponding limb. Tewaschew[26] obtained similar results after cutting the femoral nerve and the opposite effects upon stimulation of the peripheral end of the divided nerve. Taylor and Rice[25] and Mosser and Taylor[21] have uniformly obtained an increase in the temperature of an extremity following an alcoholic injection of the sciatic nerve with a strong enough solution to produce a paralysis. They

found that the minimum concentration necessary to produce paralysis produces a maximum hyperthermia.

Severance of the posterior root of the trigeminal nerve very commonly produces a vasodilatation and an increase in temperature upon the affected side. This is an observation we have made in many cases following posterior root section for the relief of trigeminal neuralgia. These results may be explained upon the work of Bayliss and Bruce, who have demonstrated the existence of vasodilator impulses which travel antidromically along a sensory nerve fiber. It would seem therefore certain that vasodilatation with its accompanying phenomena in an extremity is not entirely dependent upon the periarterial sympathetic innervation and it is probable that there is very little dependent upon any such innervation of the bloodvessel wall. On the other hand, it appears equally as definite that these phenomena are regularly obtained through the agency of both purely motor, sensory or mixed nerves of cerebrospinal origin. However, it must not be forgotten that clinical observations are on record in which removal of the vessel adventitia has produced an increase in the local temperature, hyperemia and a change in the nutrition of the tissues. After removal of the cervical and lumbar sympathetic chains in patients who have had spastic extremities, we have recorded a definite vasodilatation and hyperemia, noted particularly in the sclera of the eye and in the skin of the extremity. In addition, there has been an accompanying elevation in the surface temperature of from 1° to 1.5°. We have also recorded the fact that these effects are transient and disappear from within one to two weeks, when the normal relations are reëstablished. We have observed a hyperemia and dilatation of the vessels of the sclera and nictitating membrane in cats following removal of the superior cervical sympathetic ganglion and chain, but have found no corresponding increase in the surface temperature. The animal's furry protection may of course explain the lack or nondevelopment of a regulatory skin vasomotor mechanism. These observations should not be lost sight of and attempts must be made to supply their explanation.

For our part, we can understand how removal of the sympathetic ganglia or interruption of their rami communicantes might produce whatever physiologic results are to be found in the entire extremity. On the other hand, in view of the known anatomic

and physiologic facts, we cannot conceive of how the interruption of the minor portion of the vascular sympathetic innervation from a local segment of an artery can affect the most distal portions of the extremity.

In the consideration of vascular diseases of the extremities with symptoms and signs of deranged circulation, it is necessary to take into account the physiology and pathology of the capillary system. From the work of Hooker,[12] Krogh,[16] Rouget[23] and others it is believed that the capillaries are definitely capable of active alterations in their caliber. From this experimental work it is evident that these structures are controlled by nerve impulses and by direct mechanical and chemical stimuli. It has been shown that stimulation of the sympathetic innervation produces an active capillary constriction. On the other hand, stimulation of sensory spinal nerves results in a capillary dilatation through the mechanism of an antidromic conduction in these fibers. In a study of dermatographia, Cotton, Slade and Lewis[6] have shown the effects of mechanical irritation of the skin, which they believed were due to capillary constriction or dilatation. According to the recent views of Krogh,[16] Hooker and Dale,[13] the capillaries are influenced by chemical stimuli. It has been shown that histamine, for example, produces a marked capillary dilatation. Cannon[5] has presented evidence that in wound shock the capillaries are packed with red cells, which indicates the presence of a chemical influence.

Further, we must not lose sight of other physiologic factors which may play a very prominent part in the interpretation of circulatory diseases of the extremities. We cannot deny the influences of the frequency and force of the cardiac action, the viscosity of the blood, the quantity of the blood, the changes in the circulation of the blood under various conditions and the changes in cardiac muscle work. As a result we must recognize the apparent inadequacy of any surgical procedure in which an attempt is made to alter peripheral vascular resistance to affect materially the clinical course of such diseases.

Consequently, basing our surgical procedures entirely upon known anatomic and physiologic facts, we have attempted to record our observations in vascular diseases of the extremities after removal of the sympathetic ganglia and their rami. This principle of attack, to our mind, removes the major sympathetic

innervation to an extremity without in the least impairing the somatic nerve supply. We believe that any surgical attack upon the sympathetic system may be applied only to those diseases of the extremities in which organic lesions of the vessels cannot be demonstrated. It is illogical to attempt to produce changes in muscular contractility in the walls of vessels in which definite pathologic changes have taken place. Therefore, we should exclude thromboangiitis obliterans, arteriosclerosis, luetic arteritis and possibly scleroderma and chronic asphyxia. While many authors deny their recognition as clinical entities, we may include erythromelalgia, Raynaud's disease and acroparesthesia as diseases suitable for surgical attack. The symptomatology in these conditions has been referred to disturbances in the entire sympathetic nervous system and the importance of the relationship of the endocrine glands has been emphasized by some individuals.

Surgical Procedures. In operations upon those patients in whom the symptoms involved the upper extremities, we removed the stellate and inferior sympathetic ganglia, their branches and the intervening trunk. From one of these patients we removed the superior and middle ganglia in addition. We feel that this is quite unnecessary, since the nerves of the upper extremity receive their sympathetic fibers entirely from the stellate ganglion. In addition, we add the symptoms of a Horner's syndrome unwarrantedly. In dealing with lesions in the lower extremity we have removed the lumbar sympathetic ganglia both by a lumbar and an intraäbdominal route. We prefer the former if we desire to produce an effect in only one extremity, and the latter if we wish to remove the sympathetic supply from both extremities during one operation.

Technic of Cervical Sympathectomy. An incision is made from the inferior angle of the mandible and on the medial edge of the sternomastoid muscle downward. The line of incision crosses the sternomastoid from its medial to its lateral edge in an oblique direction to reach the superior border of the clavicle. Here the incision turns lateralward. The completed line therefore resembles a hockey stick (Fig. 1).

The fascia of the sternomastoid is exposed and the medial edge of the muscle is freed. If one desires to excise the superior cervical ganglion, the incision should be carried upward toward the tip of the mastoid process. The carotid artery, jugular vein and

vagus nerve are now in the field of operation as the medial edge of the sternomastoid muscle is retracted laterally. By gently retracting the jugular vein medially and at the same time rolling its posterior surface forward, the cervical sympathetic chain may be seen lying beneath the lateral edge of the vein on the scalenus muscle (Fig. 2). By dissecting upon the medial edge of the sternomastoid, any danger of injuring the spinal accessory nerve is avoided. The sympathetic trunk may be isolated by blunt dissection and followed as it passes laterally and downward beneath

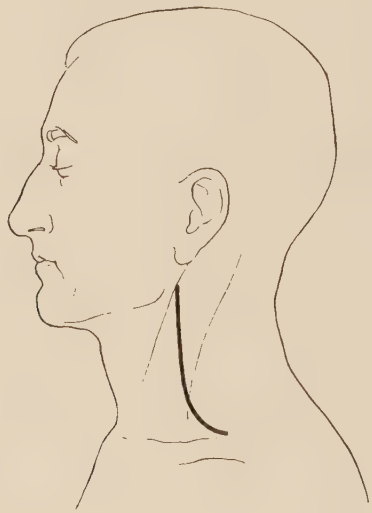

FIG. 1.—Skin incision employed to isolate and remove cervical sympathetic trunk.

the sternomastoid muscle. If any difficulty is experienced in recognizing the sympathetic trunk, one may dissect upward and identify the superior cervical ganglion and proceed from that landmark. The trunk may be divided and passed beneath the muscle and delivered into the field by gentle outward and upward traction. At the same time the lateral and inferior edge of the sternomastoid is retracted medially. In this position the inferior thyroid artery passes transversely across the field from its origin from the thyreocervical axis. The middle cervical sympathetic ganglion is usually in close association with this vessel and may

encircle it. Medial laryngeal branches, as well as the superior and middle cardiac nerves, arise from the sympathetic trunk near this ganglion (Fig. 3). The chain is then followed further inferiorly behind and below the clavicle. The apical pleura soon enters the field and should be protected. As one bluntly dissects down-

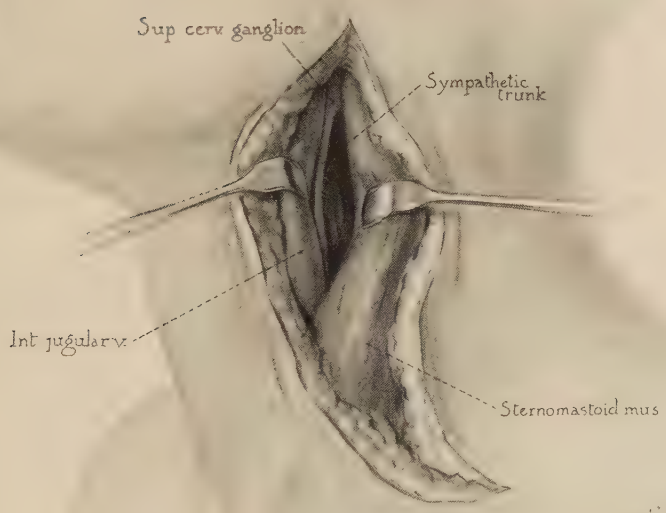

FIG. 2.—Position of cervical sympathetic trunk lying beneath the lateral edge of the internal jugular vein.

ward, the trunk must not be confused with the cardiac nerves which are almost of equal size. The parent trunk holds a more posterior position. The next structure encountered before reaching the stellate ganglion is the small inferior cervical ganglion. This may be fused with the stellate, and if separate, lies immediately superior to the latter. At this point the vertebral artery

may be seen in the field, lying medially. By exerting gentle traction upward and outward during this dissection and dividing the rami from the trunk and ganglia, the stellate ganglion may be delivered into the field from its location on the tubercle of the first rib. This maneuver makes the removal much more simple.

FIG. 3.—Relation of the middle cervical sympathetic ganglion to the inferior thyroid artery.

After dividing its rami, the sympathetic trunk is divided below the ganglion (Fig. 4).

The subcutaneous tissue and skin are closed in the usual fashion and a pad is bandaged into position to fit snugly into the supraclavicular fossa. It will be recognized that this procedure may be modified as one desires to excise the stellate ganglion only or

others of the ganglia and trunk. It is recognized that the upper extremity receives its sympathetic innervation from the stellate ganglion alone.

TECHNIC OF SYMPATHECTOMY BY THE ABDOMINAL ROUTE. A midline incision is made through the skin, fascia and peritoneum, with the umbilicus about opposite the middle point. With the

FIG. 4.—Retraction upward and laterally brings the stellate ganglion into view and its rami may be easily severed.

patient in the Trendelenburg position, the intestines are packed upward and laterally to expose clearly the posterior peritoneal wall. The aorta, vena cava and ureters may be seen through the peritoneum.

A linear incision is made through the peritoneum over the medial edge of the psoas muscle (Fig. 5). This does not expose the ureter.

The lumbar sympathetic trunk and ganglia lie upon the antero-lateral surfaces of the vertebral bodies and are partially covered by the medial edge of the psoas and on the right side by the vena cava (Fig. 6). Because of the situation of the vena cava

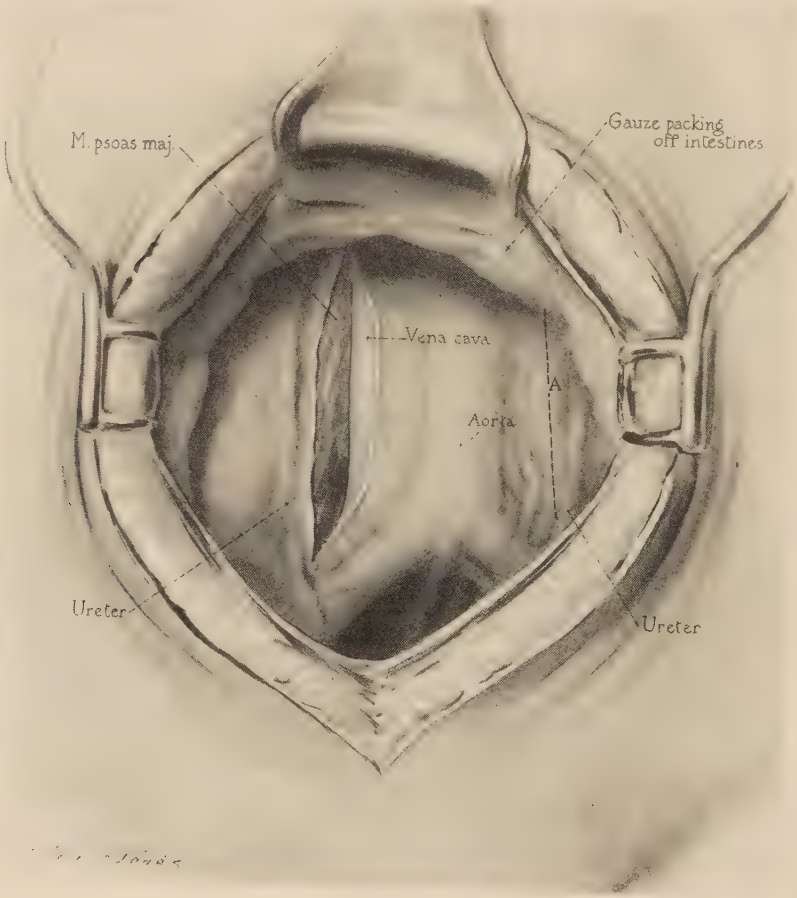

FIG. 5.—Incision through the posterior peritoneal wall over the medial edge of the psoas muscle.

the right lumbar chain is more difficult to expose. The vena cava is gently retracted medially and at the same time is lifted up. The sympathetic ganglia and trunk may be seen clearly, lying in a loose adventitious tissue. The trunk and ganglia bear a varying

relation to the lumbar arteries and veins, in that these structures may pass above or below the trunk (Fig. 7). The second lumbar ganglion usually lies just under cover of the inferior edge of the third portion of the duodenum. We have carried our excision

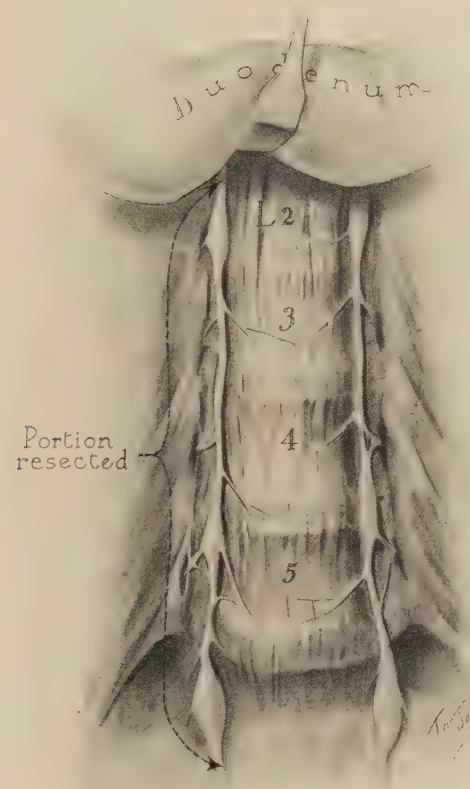

FIG. 6.—Relation of the lumbar sympathetic trunk to the bodies of the vertebra. This shows the retraction of the third portion of the duodenum necessary to expose the second lumbar ganglion.

upward to include this ganglion and have removed the chain as low as to include the fourth lumbar ganglion. This structure occasionally lies over the brim of the pelvis, and it is necessary to protect carefully the iliac vessels. Any oozing encountered is

easily controlled. We have never sutured the peritoneum, but simply allow it to fall into place. The aorta does not overlie the trunk as does the vena cava, and it is therefore much easier

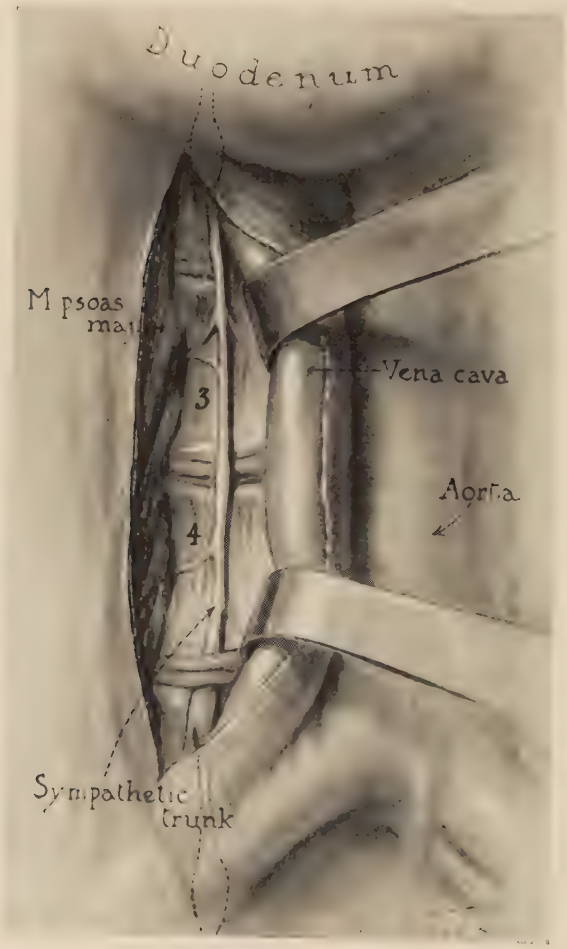

FIG. 7.—Relation of the lumbar sympathetic trunk to the psoas muscle and vena cava.

to remove the chain and ganglia upon the left side. The intestines are replaced and the wound is closed in the usual manner.

In this work we have removed the ganglia and trunk from the second to the fourth lumbar ganglia inclusive. It necessarily

follows that the communicating rami are severed as their ganglion cells of origin are removed. This abdominal route of approach permits one to interrupt the sympathetic supply to both lower extremities through the same operative wound.

Technic of Sympathectomy by the Lumbar Route. This method of approach was first devised by Royle. A complete detailed description of this operation is given by this author in *Surgery, Gynecology and Obstetrics*, 1924, xxxix, 707. We have followed this same technic and have found it to be quite satisfactory. This operation is particularly useful and is indicated if one desires to remove the sympathetic supply from one extremity only. We have found it a trifle more difficult to remove the entire chain and ganglia from the second to the fourth lumbar ganglia inclusive by this method. The procedure is also rather difficult in large, muscular or obese individuals. These small technical inconveniences are outweighed, however, by the smooth postoperative convalescence of patients who have been operated upon by this method as compared to those operated upon by the abdominal route.

Clinical Cases

Erythromelalgia: Lumbar Sympathectomy; Relief. In 1922, P. C., aged thirty-five years, a Russian-Jewish tailor, began to have an aching pain in his feet, particularly after walking. This difficulty increased in frequency and intensity so that upon his entrance into the hospital walking four or five blocks caused him great discomfort and pain. Since 1924, he had noticed that his feet, and particularly his toes, became very red and purplish in color. This was true when they were in a dependent position. This color disappeared if he elevated his feet. The redness of his toes and the dorsum of his foot was almost always confined to the area in which he experienced pain. Accompanying these symptoms, the patient also complained of swelling and a feeling of fulness in his feet. The past and family history were without significance.

Examination. The pulses of both legs and feet were full and easily palpable. *X*-ray examination of the extremities showed an entire absence of vessel-wall calcification. A marked hyperesthesia was present over the dorsal surfaces of both feet. This corresponded to the color changes which were present. It was noted that the toes and the dorsal surfaces of both feet as high as the metatarsotarsal articulations became intensely red in color if the patient allowed the

feet to become dependent. This color was also brought out by exertion or by heat. The color could be made to disappear by digital pressure, but rapidly returned when the pressure was released. Elevation of the feet caused the redness to disappear. The color changes occurred paroxysmally and were accompanied by pain.

No trophic lesions such as blebs, thickening or atrophy of the skin were seen. The nails of the second and fourth toes on the right foot were brittle and longitudinally striated. These symptoms were all symmetrical in both feet, but were more pronounced upon the right side. The remainder of the physical and neurologic examination was without note.

Blood-chemistry examination showed a nonprotein nitrogen of 45.1 mg., urea nitrogen of 14.8 mg.; creatinine 1.39 mg.; sugar of 0.127 per cent. The red blood cell count was 4,080,000; white cells, 8400; hemoglobin, 80 per cent. All urine examinations were negative.

Operation. A right lumbar sympathectomy was performed April 29, 1925. The entire right sympathetic chain from the second to the fourth lumbar ganglia inclusive was removed. The innervation to the left extremity was not disturbed.

Postoperative Course. April 30, 1925: The right lower leg is 1.5° warmer than the left. The toes are reddish-purple in color. The color change is similar to that seen when the leg is pendent, although it is now present as the patient lies in bed. No pain is present. The left foot is normal in color.

May 1: Increase in surface temperature of the right leg persists, as does the red color when the foot is supported.

May 10: Patient allowed out of bed. Right foot is still red in color, but not painful in pendent position. Left foot became red and distinctly painful after a short walk in the corridor; the right foot was not painful and the color remained the same.

May 18: Patient returned for examination. Still has increased surface temperature in the right foot, though this is distinctly less than when examined upon prior occasions. The right foot is still red in color regardless of its position. He has had no pain in this foot even after a considerable amount of exercise.

The patient returned to his work as a tailor in Rochester, New York. He has communicated with us and has been seen upon several occasions by Dr. George Whippel. The patient's report is given in abstract as to his subjective symptoms.

June 1: "My right foot is assuming its natural color and feels warmer than the left. I have had no pain in the right foot."

June 17: "There are a few little red spots on my right foot and it gives me a little burning pain. However, I am back at work."

PLATE I

Right foot of patient with erythromelalgia before and after removal of right lumbar sympathetic chain and ganglia.

June 29: "I am feeling fine. The trouble in the right foot has disappeared and I have no pain at all."

October 12: "My right foot is its natural color and I have no pain in it at all even after walking. My left foot is always cold and it gives me pain once in a while."

February 18, 1926: "My right foot is still in fine condition and feels warm to my touch. It does not change in color as it did and I have no pain in it."

The condition of this patient's foot is shown both before and after operation in Plate I. The last sketch was made in December, 1925, by Mr. N. C. Jacobs, of the Department of Anatomy, University of Rochester Medical School.

The most striking symptoms in this case were the redness and pain occurring in paroxysmal attacks and influenced by posture, heat and exertion. These symptoms were localized symmetrically in the distal portions of the extremities, although the right foot was more seriously affected. There was an absence of all evidence of organic vascular disease. This undoubtedly was a case of erythromelalgia. The arterial pulsations, paroxysmal attacks and absence of intermittent claudication differentiate it from thromboangiitis obliterans, with which it may be confused. Cassirier has divided this condition into two types: (1) That in which the local symptoms follow the paths of certain nerves and (2) that in which the distribution is diffuse over various peripheral parts of the extremities. In the first group, he states that an irritative process in the peripheral nerves exists with predilection for the vasodilator and secretory fibers. In the second group he believes that a central nerve lesion is present and that in all probability the sympathetic system is the seat of the disease. No pathologic evidence is offered for this latter statement.

There seems to be no doubt that after a year the patient's condition is definitely improved. We expect to operate upon the other extremity at the patient's convenience, but at this time it serves as an excellent control for the experiment.

Raynaud's Disease: Right Cervical Sympathectomy; Relief. E. W. aged twenty-eight years, a Polish housewife, first noted in 1918 that the middle finger on her right hand would become pale and blue in color when she was out in the cold weather or when she had her hands in cold water. This color change was always accompanied by severe

pain and numbness and tingling in the finger. During the following six years these symptoms gradually progressed to involve the other fingers and the thumb of the right hand. The patient then noticed that excitement or worry would produce the same symptoms and under such conditions her face would become deeply flushed. In 1924, this same group of symptoms appeared in the fingers of the left hand. The pain in her hands had been very severe and she had sought relief in many ways without success.

The patient also stated that she very commonly had ulcerated areas on the ends of her fingers which followed the slightest trauma. The healing of these lesions was always followed by retracted scars. During the past year the patient had been complaining of pain in the toes of both feet, accompanied by a slight bluish discoloration. The past history was without note and no relation could be drawn with her present condition.

Examination. The patient's face is of a dark reddish-purple color, particularly when she is under observation. Her skin is naturally dark in color. There is a bilateral exophthalmos. The thyroid gland is palpable but not abnormally enlarged. There are no bruits or thrills over it. Blood-pressure is 120/68; pulse, 88.

By placing the hands in cold water or by surprising the patient an attack of discoloration of the fingers can be produced. Normally the hands are perhaps slightly redder in color than those of the average individual. During an attack the color rapidly changes to a bluish-gray. This involves all of the fingers and thumb down to the metacarpophalangeal joints, except the fifth fingers which become discolored only over the distal phalanges. During this attack the radial pulse is full and strong. The fingers feel cold and clammy. They are very tender to the slightest pressure, particularly over the extremities. The tips of the fingers and thumbs are hard and boardlike, and are very painful at all times but more so during an attack. The skin in these areas is scaly and indurated. The tip of the right index-finger is ulcerated. The end of the distal phalanx is exposed. It has been in this condition for five weeks and resulted from a rather insignificant trauma. All of the peripheral pulses could be palpated with ease.

A tourniquet was applied to both arms, but no hyperemia could be produced after its release. *X*-ray plates of the arms showed no evidence of vessel calcification. *X*-ray plates of the hands did not show an atrophy in the tips of the distal phalanges characteristic of Raynaud's disease in the later stages.

The basal metabolic rate of the patient upon several occasions was + 12. Blood-chemistry examination showed nonprotein nitrogen of 27.4 mg.; uric acid, 4.24 mg.; creatinine, 1.5 mg., and sugar 0.11 per cent. The blood and urine examinations were normal.

PLATE II

Right and left hands of patient with Raynaud's disease after removal of right cervical sympathetic chain and the right stellate ganglion. Sketched during an attack of local syncope and asphyxia.

Operation. On January 29, 1926, a right cervical sympathectomy was performed. Since we were anxious to leave no possible sympathetic innervation to the upper extremity from the cervical chain, we removed the entire chain and all of the ganglia from the superior to the stellate inclusive. Naturally, all of the rami of the latter were severed. Again, we will simply remove the stellate ganglion because we feel that this structure contains all of the cells of origin of fibers innervating the upper extremity.

Postoperative Course. The patient made an uneventful recovery from her operation. No sensory findings were present about the face or neck. We may state here that in none of our cases have we found any subjective or objective sensory disturbances in the face or neck following a sympathectomy. There is an enophthalmos, small pupil, narrow palpebral fissure, and the scleral vessels are dilated upon the right side. It will be remembered that the patient had an indolent ulcer on the index-finger of her right hand when she came into the hospital. We removed the dressing and simply allowed the finger to be exposed without in any way treating the lesion.

February 4, 1926: The patient has had no pain and no attacks of local syncope and asphyxia since her operation. The right index-finger is entirely healed and the skin of the tip of the finger is pink. The right upper extremity has been from one to two degrees warmer than the left since her operation. The patient states that the examiner's hands are colder when felt with her right hand as compared with her left. No difference in the color of the fingers or hands can be detected except during an attack.

February 7: The temperature difference is less today as measured with a surface thermometer. The patient's hands were immersed in ice-cold water today. After fifteen minutes no change had occurred in either hand. As soon as they were brought into the air the attack of syncope and asphyxia began. The change in color to the typical chalky bluish-white blanching occurred quickly in both hands. However, the extent of the color change and the degree of change were quite different in the right hand. The ends of the thumb and fingers, except the right fifth finger, were involved only over the middle and distal phalanges as contrasted to the condition in the left hand in which the changes extended into the palm of the hand. Further, the return to the normal color of the right hand was completed five minutes before that in the left.

March 4: The patient was taken to the artist for a colored sketch of her hands. Before entering the presence of this stranger, her hands were normal in color. Immediately upon meeting him she began to develop an attack of local syncope and asphyxia. The result of this attack which lasted thirty minutes is seen in Plate I. She returned

upon several occasions in order that this color might be verified and it was not until the third visit that the change did not occur and cold-water immersion was necessary.

March 21: The patient notes a great difference in the condition of her right hand. She has had no paresthesia or pain and her hand never feels as cold as the left nor as it did previously. The lesion of the right index finger has never recurred. The Horner syndrome on the right side has remained unchanged.

April 6: The patient has had no pain in her right hand. During an attack, the middle finger of the right hand becomes cyanotic from the tip to the proximal interphalangeal joint while the rest of the fingers and hand are normal in color. The left hand becomes markedly blue and painful. There has been a decided improvement since the time represented in Plate II.

The occurrence of induration in the tips of this patient's fingers suggests, of course, scleroderma. Since vasomotor changes may be early manifestations of scleroderma this condition may be difficult to differentiate from Raynaud's disease. Buerger quotes Cassirier as grouping Raynaud's disease into three types: The typical cases show symmetrical gangrene, dystrophic changes in the distal portions of the extremities, with thickening of the skin and immobilization of the tendon sheaths and joints. The second group comprises the cases in which local asphyxia and syncope initiate the disease. There is no progression into a state of gangrene, but on the other hand a chronic stage of vasomotor, sensory and sclerodermic symptoms occurs. The last group consists of rare cases which show vasomotor symptoms in the presence of an advanced scleroderma. Our case obviously belongs to the second group. Certainly it belongs to the group of vasomotor neuroses of which the Raynaud syndrome is an example. Buerger[3] believes that in these cases there is an irritative or perhaps a paralytic process in the vasomotor system. Cassirier believes that the vasoconstrictors are in a condition of increased irritability.

Adson and Brown[1] have reported a case of Raynaud's disease in which they removed the lumbar sympathetic chain and stripped the adventitia from the external iliac vessel upon the same side. They have reported a marked improvement in the extremity upon the side operated upon. They believe that such a double procedure prolongs the physiologic effects of removal of the sympathetic innervation.

While time has not elapsed following the operation equal to that in the first instance, we feel that this patient has received definite improvement and relief.

We have operated upon another patient with Raynaud's disease and removed the stellate ganglion upon the right side. This patient suffered from local syncope and asphyxia in all four extremities but the symptoms were more advanced in the hands. Thickening of the skin and immobilization of the tendon sheaths and joints of the hands were present. This patient was operated upon so recently that we have had no opportunity to determine accurately just what her postoperative status is. Consequently we refrain from including a detailed history of her case.

Thromboangiitis Obliterans: Lumbar Sympathectomy; No Relief. Observation of a Second Case after Periarterial Sympathectomy Elsewhere; No Relief. We also wish to record our experience with sympathectomy in this disease and because of the clinical failure we refrain from giving a detailed report.

We removed the right lumbar sympathetic trunk and ganglia from a patient whom we believed had an erythromelalgia. The red color of this man's extremities was intensified in the dependent position, but a fact to which we did not give sufficient weight was that this color was not paroxysmal but was constantly present. He had a trophic ulcer upon the end of his great toe on the right foot and all but his dorsalis pedis vessels pulsated. *X*-ray plates of the extremity failed to show any calcification in the vessels. The pain persisted, as did the color changes in the foot, and after two months no improvement whatever was evident. Because of the intense pain which persisted and because of an advancing gangrene, an amputation was performed. Examination of the vessels showed the organic changes of a thromboangiitis obliterans.

We have observed a second example of this disease which was verified by an examination following amputation of the extremity. A periarterial sympathectomy had been performed elsewhere nine months previously. The patient complained bitterly of pain in his foot and required morphine for relief. The color changes in his toes and foot were typical of those seen in the preceding case. However, no gangrene had developed, although no pulsation could be felt in any of the peripheral vessels.

We made a diagnostic error in the first of these cases of thromboangiitis obliterans. Recognition of the lesion and a thorough understanding of the organic changes in the vessel wall are suffi-

cient contraindications to an operation designed to attack the sympathetic innervation of an extremity. One cannot hope to dilate or constrict an arterial wall so altered by a pathologic process such as Buerger has so carefully described in this disease.

Discussion. In discussing the physiologic effect of removal of the sympathetic innervation to an extremity, one is prone to go beyond the few known facts in order to furnish a plausible and logical explanation.

First of all, we know that arterial decortication, or removal of the sympathetic chain, produces hyperemia and increased temperature in the corresponding extremity in man. Second, the symptoms of paralysis of the vasoconstrictors or unfettered influence of the vasodilators gradually disappear within from one to two weeks after the operation. Third, the sympathetic innervation to the bloodvessels of an extremity is furnished at segmental intervals by way of the spinal nerves. Fourth, that any fibers of sympathetic origin within the bloodvessel wall are not continuous along the wall of that vessel. Fifth, in certain vascular diseases of the extremities characterized by paroxysmal vasomotor symptoms and the absence of organic vessel pathology, removal of the sympathetic chain is followed by an improvement in symptoms. Finally, we must bear in mind the fact that there are many variable factors concerned in the control of the peripheral circulation, of which the vessel musculature and caliber are but a part.

The known anatomic and physiologic facts concerning the origin, distribution and supply of the sympathetic fibers to the extremities explain the results obtained upon interrupting such an innervation at its origin or in its course. Thus, removal of the somatic nerve supply produces definite physiologic effects in the corresponding extremity. Such procedures and results are logical and in the event of interruption of the sympathetic chain are the result of removal of the active vasoconstrictor mechanism. Since the sympathetic innervation to the vessels of an extremity is supplied in a segmental manner, *a priori* the removal of a small portion of the terminations of these sympathetic fibers could not be expected to affect permanently any portion of the vessel except the local segment operated upon; on the other hand, both local and distant favorable effects in the extremity after cortical decortication are reported. The effect of such a procedure upon the

vessel caliber at the site of operation might very well be explained upon the basis of the interruption of a local short reflex mechanism within the wall of the vessel. Our knowledge makes it impossible to explain accurately the hyperemia produced in the foot by removal of the adventitia upon a small segment of the femoral artery.

While it would seem more logical to remove the sympathetic ganglia and rami rather than to decorticate an artery, we are still met with the inability to explain completely our results in cases of vascular disease of the extremities. Primarily we do not know the underlying pathology in these cases of vascular disease which would appear to be benefited by removal of the sympathetic innervation. It is assumed that those diseases in which no organic vessel pathology can be demonstrated are the result of a vasomotor neurosis and that the sympathetic innervation to the extremity is at fault. There are no facts to support such a contention except the empirical observation that removal of the sympathetic innervation effects an improvement. In view of the many circulatory factors previously pointed out which may have an important relation to these diseases, we cannot convince ourselves that an alteration in one of the peripheral mechanisms may be the sole key to the situation. This doubt is somewhat strengthened by the observation that the physiologic results of interruption of the sympathetic innervation gradually become less pronounced and finally disappear. There is abundant experimental support for this clinical observation. Just what the actual mechanism of this readjustment is we are at present unable to say.

Conclusions. 1. The peripheral bloodvessels receive their sympathetic innervation in a segmental manner by way of the somatic nerves.

2. Removal of the sympathetic innervation to an extremity in man is followed by hyperemia and hyperthermia, which gradually disappear within one to two weeks following operation.

3. In certain vascular diseases of the extremities characterized by paroxysmal vasomotor symptoms and the absence of organic vessel pathology, removal of the sympathetic innervation is followed by an improvement in symptoms. Among this group we may include erythromelalgia and Raynaud's disease.

4. There are many physiologic factors concerned in the control

of the peripheral circulation, of which the vessel musculature and caliber are but a part.

5. In the present state of our knowledge concerning the pathology of the group of vascular diseases known as vasomotor neuroses, we are unable to explain completely the effects produced by removal of the sympathetic innervation to the extremity.

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DISCUSSION

Dr. W. J. Mixter, Boston, Mass.:

Dr. Kanavel's paper opens up the field of the application of the Hunter-Royle operation in the vascular lesions. It has seemed to me that probably that field was of greater value under our present knowledge than the field of the spastic paraplegias. So far I have not used it in the vascular lesions, but I have noted in one case a very considerable increase in temperature on the side so operated upon in a patient who was operated upon for spastic paraplegia. It is now some sixteen months since he was operated upon, and his foot on the operated side is definitely warmer than his foot on the side which was not touched.

Dr. George P. Müller, Philadelphia:

I have performed operations upon about 67 cases of periarterial sympathectomy, and I agree with Dr. Kanavel when he says that the operation is contraindicated in cases of Buerger's disease, senile and diabetic gangrene and luetic arteriosclerosis, although occasionally in Buerger's disease we have had remarkable amelioration of pain, which is, in a way, what the patient is most interested in. You cannot possibly hope to effect any changes in an artery which is completely obliterated.

On the other hand, Buerger says that the disease involves only the larger vessels, that the capillaries escape. One would think, reasoning from Krogh, that the sympathetic innervation would go all the way to the Roguet cells of the capillaries, but it does not seem to do any good to get an innervation in the capillary bed, because we only increase the stasis if the artery is unable to push the blood through fast enough.

We have had about a dozen apparent cures. These will be reported in the *Archives of Neurology*, probably in the fall. Among them are 2 cases of Raynaud's disease, 1 in a rather old man who was well six months and then was lost sight of. The other patient, a woman, has remained well for something over two years. It seems to me that periarterial sympathectomy might be tried first before the more extensive procedure of opening the loin or the abdomen in order to cut the gray rami.

Dr. Edward W. Archibald, Montreal, Canada:

Of course this whole subject is full of interest as being full of the unknown. At the present time there is still discussion going on as to whether the sympathetic supply to the arteries is segmental entirely, being given off at successive levels from the somatic nerves, or longi-

tudinally, that is, running the whole length of the artery and given off from high up. The balance of opinion favors the segmental theory.

I might record one observation which speaks for the bulk of the supply being segmental. The patient had had constant pain in the right foot following a war injury at the level of the fifth lumbar vertebra. I did a sympathectomy upon, first, the right external iliac, and later the right common iliac, going up to the bifurcation of the aorta. On these two occasions, after stripping 2 to 2½ inches of the vessel, I got no response whatever locally in the artery in the way of constriction, nor any particular change in the warmth of the affected foot. I concluded that at least for those segments there was no particular nerve supply, such as Leriche indicates, and that therefore the supply was probably segmental, the artery being left unsupplied in these and perhaps in other segments. So much for vasomotor effects!

On the other hand, it is being generally recognized that the arterial sympathetic plexus carries pain fibers. It is growing anyhow increasing obvious to me that the relief of pain brought about by a sympathectomy in various types of cases—including, as Dr. Müller has indicated, the Buerger type, as also pure arteriosclerosis—is frequently extraordinary. I rather think that the future of this operation will hold for us much more relief from painful conditions than from ulcerative conditions or angiospastic conditions. As an illustration I might relate the case of an old man whose leg was amputated above the knee for arteriosclerotic gangrene, following which there remained terrific spasms of pain in the stump in addition to the ordinary reflected pain in the amputated foot. I finally did a periarterial sympathectomy on the femoral. The artery was absolutely closed, thrombosed and atheromatous. There could be no possible effect on the caliber of the artery nor on the circulation in the leg. The relief of pain was immediate and permanent.

Dr. Kanavel (in closing):

Concerning the question of the cure of these various diseases, we wish to emphasize that we realize and believe that there are other factors besides involvement of the sympathetic nervous system, and that until we have evaluated the entire group of factors dealing with these diseases we are unable to say that this or that particular operation can or will cure the disease.

We do not say that periarterial sympathectomy should not be done. By no means do we feel that we have solved the problem. We have only stated the results of our experimental investigation, our clinical observation and study of the literature. We do believe this operation has a scientific basis; we recognize that many satisfactory clinical results have been obtained by periarterial sympathectomy.

ADVANTAGES OF THE PRIMARY SUPERIOR POLAR ATTACK IN THE REMOVAL OF SUBSTERNAL THYROIDS

By DONALD GUTHRIE, M.D.
SAYRE, PA.

The anatomical relations and the mechanical factors which are responsible for the production and development of substernal or intrathoracic goiter have been beautifully explained by Pemberton and Lahey.

It is in the removal of these substernal masses, often of great size, in a patient who has severe mechanical difficulty with respiration, that the surgeon gets his worst thrills, for tense situations are frequently wont to arise.

Pemberton, in 1920, suggested that the operation begin by a mobilization of the upper superior pole on the side which contains the substernal growth, in contradistinction to the accepted and generally employed method of primary elevation of the substernal mass, and stated that this primary mobilization and elevation of the superior pole isthmus and visible part of the thyroid would usually cause the hidden mass to roll out of the mediastinum without undue traction or vain efforts to dislodge it—the likelihood of injury to the trachea and the recurrent laryngeal nerve, the need for immediate tracheotomy and the dangers of deep and concealed hemorrhage all being reduced to a minimum.

It is most important to obtain the best exposure of the thyroid bed at the beginning of the operation. The low collar incision should be long and the skin and platysma muscle reflected well back on both sides (Fig. 1). All bleeding should be controlled at this step, as it is important to get rid of all clamps. The sternohyoid muscles on both sides are best clamped and divided, and part of the sternomastoid may be cut if the exposure is not adequate. Free separation of the thyrohyoid muscle from the gland is important also (Fig. 2).

If cervical lobes are very large it may be best to resect the lobe on the opposite side to the substernal mass first, in order to secure additional space before the elevation of the offending lobe. This, however, is rarely necessary. The trachea should be located at once, and a method for its rapid exposure planned should the

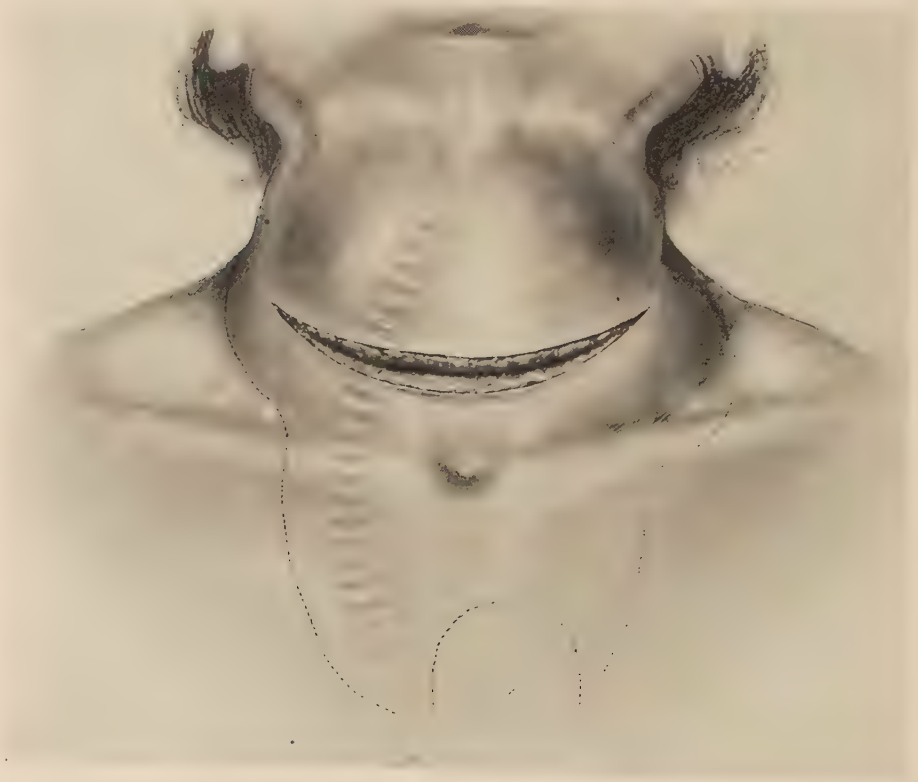

FIG. 1.—Collar incision. Drawing showing substernal enlargement with deformity of trachea.

respiratory difficulty become embarrassed enough during this early attack upon the thyroid to warrant a tracheotomy. The upper pole is sought, severed between clamps and ligated at once. The attack then extends to the lateral thyroid veins, which are clamped and ligated, for it is unwise to allow many clamps to collect in the wound, as their weight may increase respiratory

difficulty to a dangerous degree. With this amount of mobilization, the upper pole is elevated and drawn gently downward from without inward or in a reverse manner, the manipulations of the surgeon being guided by the respiration of the patient and the helpful coöperation of an experienced anesthetist. In the

FIG. 2.—Unilateral section of muscles. Muscles are to be sectioned on opposite side.

dissection at this stage it is well to leave small amounts of thyroid tissue on top of the trachea and at the side for protection of the trachea itself and the recurrent laryngeal nerve and the parathyroids. A clean fascial dissection in this area is fraught with grave dangers (Figs. 3 and 4).

The ascending branches of the inferior thyroid artery are next

encountered, and after these are severed a wide degree of mobility of the gland will be found possible. In fact, very often a large substernal mass may be elevated into the wound by the most gentle traction, occupying now the space above made vacant by the mobilized and elevated thyroid. All that is necessary now is to ligate the inferior thyroid pole and remove the gland (Fig. 5).

FIG. 3.—Relation of recurrent laryngeal nerve to trachea and esophagus. Anatomical dissection.

It is not possible to employ the method herein described in the removal of all substernal thyroids. Many will have to be pried out of their beds hurriedly, others removed by morcellation; cysts will have to be ruptured to permit rapid removal; but many of these substernal masses can be easily removed in this way. When one compares this technic with the one of rapid elevation, attended

so often by an increase in respiratory difficulties instead of respiratory improvement, the rapid and vain search for a hidden and deformed trachea in a neck already jammed with thyroid before the elevation of more thyroid, the danger of further stretching an already overstretched recurrent laryngeal nerve, the

FIG. 4.—Relation of recurrent laryngeal nerve to trachea and esophagus. Anatomical dissection.

danger of deep and alarming hemorrhage due to pulling off the inferior thyroid vein or artery or both, and the liability to injury to the recurrent laryngeal nerve, the internal jugular vein or the parathyroids, as a result of the hectic efforts of the surgeon to control this severe and alarming hemorrhage, the advantages of the method are obvious.

Since employing this operation we have not had the need for tracheotomy, and we have been impressed by the lessened post-operative respiratory and voice difficulties in our work.

In a paper, in 1918, on the "Temporary Loss of Voice following Thyroidectomy," the writer urged that the trachea and larynx be

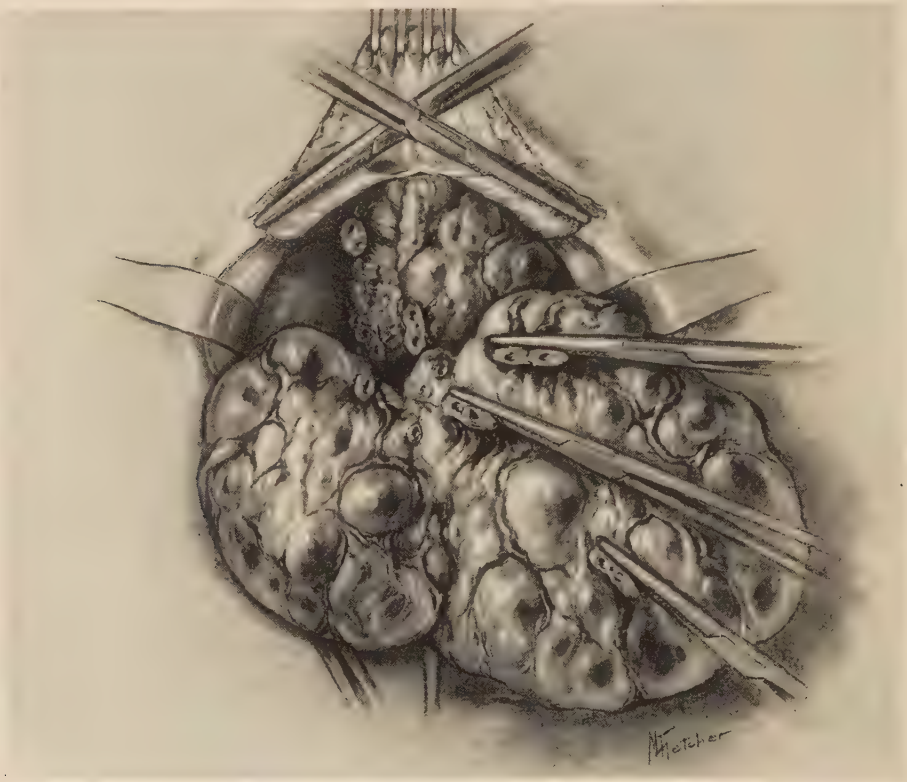

FIG. 5.—Primary mobilization of right superior pole. Right lobe and isthmus freed from trachea. Note substernal mass presenting into wound. Left lobe undisturbed.

protected in all manipulations which would cause any change in the respirations of the patient during the operation for the removal of goiter. An everchanging attack was suggested, which, with the coöperation of the anesthetist, was planned to proceed in any direction which was compatible with quiet respiration on the part of the patient, and the direction of the attack be changed at once

with the development of the slightest change in the patient's respiration.

A clean dissection of the trachea was strongly advised against, because of the dangers of postoperative voice difficulties, due to tracheitis, which follow irritations from such dissections, and it was urged that small amounts of thyroid tissue be left on top of the trachea and along its sides as a protection to the trachea itself and recurrent laryngeal nerve.

Pemberton contends that local anesthesia is safer than general anesthesia in this type of work, because so many of these patients learn to employ the accessory respiratory muscles as respiratory difficulties increase and that a general anesthetic destroys the coöperative action of these accessory muscles. We cannot quite agree with Pemberton on this point, because we have always employed general anesthesia in our work and we have noticed no additional respiratory difficulties upon anesthetizing these patients or at the beginning of these operations.

The use of ethylene-and-oxygen anesthesia is especially recommended in thyroid surgery. The absence of mucus and the quiet respiration make it extremely safe, for any disturbance with respiration during an operation for goiter may be charged to the surgeon's manipulation and not to the anesthetic.

This excellent and valuable method of Pemberton's has not been generally adopted, and the writer, who has employed it for five years in his work, and realizes full well its value, wishes to call attention to it and urge its more general adoption.

DISCUSSION

Dr. Charles H. Frazier, Philadelphia:

I should like to ask a question which might seem to be impertinent or not because of my limited experience. With regard to the removal of very large thyroid masses from the neck, it is recorded in the literature a number of times that there have been occasions in which alarming respiratory attacks have necessitated the introduction of a tracheotomy tube to save the patient's life, and that this respiratory attack is alleged to be due to the collapse of the trachea. On the basis of that statement, in the Thyroid Clinic at the University Hospital we have always included among the instruments for thyroidectomy a tracheotomy tube, but up to the present time it has not been used. I am rather of the belief that the observations previously made were mistaken to this extent, namely, that the respiratory difficulty is not due to the collapse of the trachea, but is due to a bilateral trauma to the recurrent laryngeal nerves.

Dr. Guthrie (in closing):

I have never considered the need for tracheotomy during the operation to be due to double injury to the recurrent laryngeal nerves, as suggested by Dr. Frazier. I have never had to do a tracheotomy upon a patient upon the table, but I have been obliged to perform tracheotomies upon four patients during the first twenty-four hours following operation. I am more inclined to agree with Dr. Lahey, that this need for tracheotomy was due to postoperative swelling in an already narrowed trachea or to plugs of mucus which follow the trauma of removing the thyroid.

I am sorry Dr. Lahey did not discuss this method of removing substernal thyroids, because I should have been glad to find out whether or not he agrees with this attack. Since we have used it we have had far less trouble with these patients upon the table and far less reaction postoperatively. It is astonishing oftentimes to see what large masses will present themselves in the wound that has been made open by the superior polar mobilization of the thyroid.

THE MIDLINE BLOODLESS APPROACH TO THE POSTERIOR FOSSA

BY CHARLES H. FRAZIER, M.D.
PHILADELPHIA.

TAKING it all in all, operations for the exposure of lesions below the tentorium cerebelli are approached with greater anxiety than those for lesions above the tentorium. Obviously there are many reasons why this should be so, chief of which is the proximity of the field of operation, and of the lesion itself, to the medulla oblongata with its centers presiding over the blood-pressure, the cardiac and the respiratory mechanism. The functions of these centers is sometimes already impaired by increased intracranial pressure, so characteristic of posterior fossa lesions. The respiratory rate may already have been retarded, the pulse-rate often increased and the blood-pressure raised. Or it may be that Nature has been able to compensate for the increased pressure, with its tendency to impair the circulation of the medulla, by a compensatory increase in blood-pressure. At all events, before the operation there is a delicately balanced mechanism, safeguarding the function of these vital centers, and any procedure suddenly disturbing the balance will soon show its effects. How rapidly the pulse-rate may increase in the induction of ether anesthesia alone, before the incision is made or in the early stages of an operation performed under local anesthesia! So that when the cerebellar exploration is staged there is in the air of the operating room a sense of tension, of apprehension and, possibly, anxiety.

But in addition to the fact that the operative field is in close proximity to the medullary centers, there are other factors which may be regarded as obstacles peculiar to the posterior fossa exploration. The anatomic structures and relationships of themselves are hindrances to an unrestricted exposure of the lesion. The medulla oblongata below, the lateral sinuses above, the mastoid

processes to either side, are boundary marks, within which all manipulation must be conducted. Altogether, the "cerebellar" exposure offers a striking contrast to the unlimited exposure of either cerebral hemisphere, so readily effected by the reflection of the cranioplastic flap. Admitting as we should the greater risk of the "cerebellar" patient, every means should be adopted to safeguard the operation. Many precautionary measures may suggest themselves to the readers, but I wish to discuss more particularly only three—the anesthetic, the two-stage operation and the method of exposure. It must be assumed that when the

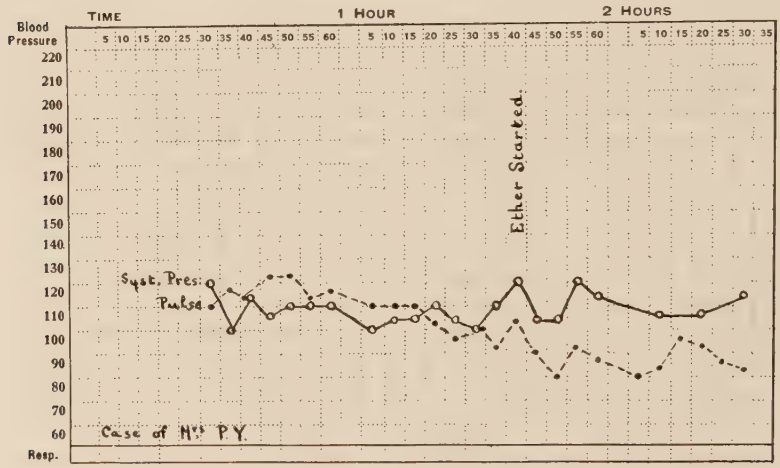

FIG. 1.—Blood-pressure and pulse chart. Note the decline of the pulse-rate under light ether anesthesia.

lesion is exposed the judgment of the operator must control the subsequent steps, whether the lesion, in the first place, be operable and, if operable, how with the greatest safety and effectiveness it may be removed.

1. THE ANESTHETIC. Whatever preference the operator may have or whatever prejudices, there can be no doubt that all intracranial explorations, and more particularly those of the posterior fossa, can be conducted with greater facility and with immeasurably greater safety under local anesthesia. At one time I was wont to think that childhood might offer certain restrictions to the use of local anesthesia, but it is surprising how tolerant even

the child is to this somewhat prolonged and tedious ordeal. Throughout an operation that from beginning to end may require two or two and a half hours, even a child of five years may be kept quietly composed while the various steps of the operation are in process of performance.

Local anesthesia is induced with a solution of 0.5 per cent novocaine, to which has been added 3 drops of a 1 to 1000 solution of adrenalin chloride to the ounce of novocaine solution. How effective the adrenalin solution may be in prolonging the influence of the novocaine in helping to control bleeding is quite problematical. The injection under pressure of the novocaine solution in abundant quantities of itself is sufficient to cause a tissue anemia which, with the aid of digital pressure, renders the incision bloodless until the hemostats have been applied and the margins of the wound everted. The system of applying the hemostats assures perfect control of bleeding from all sources above the level of the aponeurotic layer until the operation is concluded. As one approaches the muscle mass below the aponeurosis and the pericranium, additional injections of the novocaine solution should precede the division of the muscles and the separation of their attachment to the superior and inferior curved lines and to the surface of the occipital bone itself. Thus, each succeeding step until the bone is reached is rendered painless. The penetration and the removal of the bone, while always somewhat disturbing to the sensibilities of the patient, of themselves are painless procedures. The psychic effect of the crunching sound of the rongeur forceps may provoke a groan of protest, but the surgeon knows his patient is not suffering because of pain.

From this point on, including the ligation of the occipital sinus, the free incisions in the dura and the subsequent steps incidental to the exposure and removal of the tumor, the operation continues painlessly. For its conclusion there remains the closure of the wound, and here we have discovered to the satisfaction and well-being of the patient that the operation should be concluded, paradoxical as it may seem, under the influence of ether narcosis. Unquestionably in the course of the one and a half to two hours that may have transpired already, the patient has been under a degree of nervous tension in maintaining quietness of body and controlling any emotional expression. At this juncture there is a state of nervous or emotional fatigue, and the patient

may easily be intolerant to further insult, such as the introduction of a hundred or more sutures. To put the patient in a state of complete relaxation of mind and body and to avoid the discomforts and the nagging effect of needle punctures, for by this time the effect of the local anesthesia has worn off, we resort to the use of ether narcosis. If this term or recommendation were used without qualification one might be justified in making protest: "Why, when the operation is practically completed, add to what the patient has already endured, the acknowledged disadvantages of a general anesthetic?" To this we would reply that we have found with the use of an extraordinarily small amount of ether, perhaps $\frac{1}{2}$ ounce, a state of analgesia and semiconsciousness may be obtained, most welcome to the patient, ensuring quietness of mind and freedom from pain until the wound layer by layer is closed with the care which must be taken to ensure against a cerebrospinal leak. This "ether nap," if we may call it so, at the concluding stage of the operation we have found a most beneficient addition to the performance of all cranial explorations, whether it be of the cerebellar or cerebral hemispheres, and we endorse it with every assurance that it not only does not add to the burden to be borne, but lightens it at a time when the maximum of tolerance on the part of the conscious patient has been reached. We have often seen the pulse-rate, that may have increased measurably, slowly decline after the administration of ether has been begun and the patient, without struggling, drops off to sleep. (See Fig. 1 and Fig. 2.)

The obvious retort to this plea for employment of ether in the final stage of the operation might be this: If ether is to be used at all why not use it throughout? Perhaps at the beginning of this discussion of the problem of anesthesia the disadvantages of ether anesthesia in all incranial explorations might have been stated. It was assumed that these are generally recognized, but should there be any doubt in the mind of the reader, it may be stated here in no uncertain terms, that general ether narcosis adds to the risk of operation. Unquestionably there is more bleeding from various channels; unquestionably, because of greater venous congestion, the tension within the cerebellar mass is greater; unquestionably the vomiting of postether narcosis is disadvantageous. Much could be said in amplification of these reasons, dogmatically stated, but should there be any doubt as to

their justification, experience at the operating table will furnish conclusive and convincing proof. Perhaps it may be thought by some that the discussion of the anesthesia problem has been unnecessarily drawn out, but after all it must be admitted that only by attention to each detail in any complicated surgical procedure can the pitfalls be avoided and the hazards be held to the irreducible minimum.

2. The Two-stage Operation. The two-stage operation at first thought seems an acknowledgment of incompetence and unpreparedness. Certainly it should not be advised as a routine

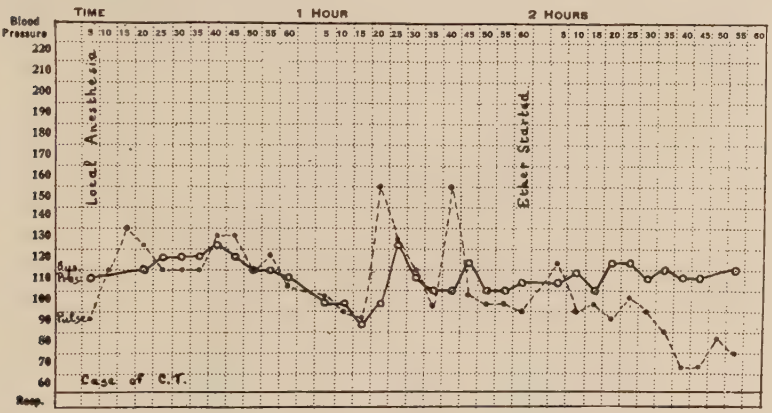

Fig. 2.—Blood-pressure chart and pulse chart during an operation upon a patient with a cerebellopontile angle tumor. Note (1) the comparative uniformity of the systolic pressure throughout the operation; (2) note a declining pulse-rate after ether was started. Pulse at the beginning of the operation 90, at the close 75.

practice, for it has many disadvantages. It prolongs the number of hospital days; it lowers somewhat the patient's morale, when told after the first stage there must be another before the tumor can be removed; there are greater possibilities of complications in wound repair when operating in a field that has recently been traumatized and robbed of some of its normal blood supply. But in the interests of safety in this field as in others, we must recognize the limitations of the human mechanism, its tolerance to insult and the endurance of the individual patient. Periods of excessive vomiting may have interfered with nutrition, the unavoidable employment of dehydrating agents and the restric-

tion of fluid intake may have reduced the blood volume and otherwise disturbed the proper balance of fluid requirements. From every point of view the condition of the patient may be anything but fit for operation, much less a formidable one. Should there be an operable tumor the chances of recovery after its removal

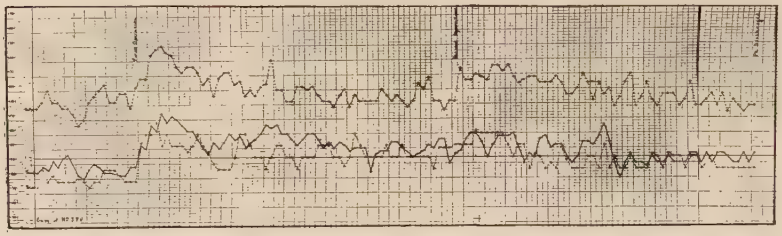

FIG. 3.—Temperature, pulse and respiration from the time of patient's admission to discharge. Following the first operation, extraordinary improvement: headache, vomiting and vertigo relieved; nutrition improved; transformation to a good operative risk.

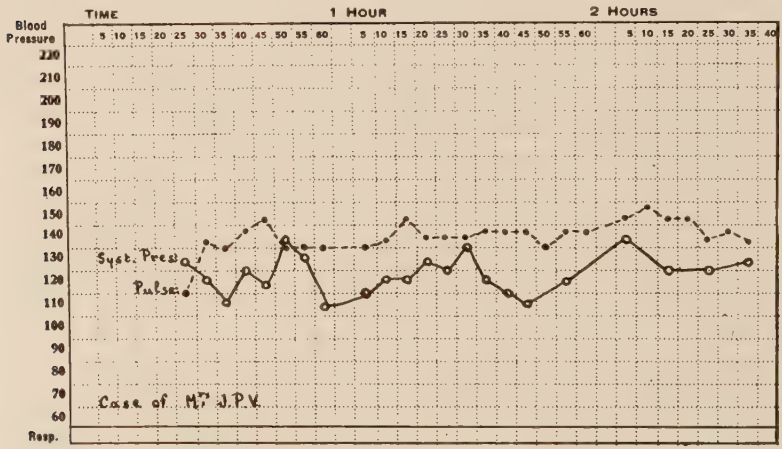

FIG. 3*a*.—Systolic blood-pressure chart. Note uniformity of systolic pressure throughout second stage.

are many times greater if opportunity be afforded for transforming what is admittedly a poor operative risk to one that is as nearly desirable as conditions may permit. A bedridden, dehydrated, ill-nourished and more or less demoralized subject by preparatory measures may be converted into an ambulant one, properly nourished and with a normal fluid intake and output.

How much time should elapse between the first and second operation is a question with which we are invariably confronted. The answer to this depends, of course, upon the circumstances peculiar to the individual case. Generally speaking, the shorter the interval the better. Forty-eight hours is better than a week,

FIG. 4.—Photograph of tumor removed in case charted in Fig. 3.

but as a rule so little can be accomplished in the shorter period that in practice longer periods must intervene. Consequently, we find one or two weeks, perhaps more, may have elapsed before we are quite content to make the final effort. We would not want to leave the impression that the two-stage operation is the

rule, rather the exception. As a matter of fact, it is only in the exceptional case that such is our practice. It is the recognition of its usefulness and helpfulness in the selected case that warrants mention.

Technic of Suboccipital Exposure. This operation has passed through several developmental stages. At first we were wont to make only a curved incision from mastoid to mastoid, with the apex of the curve above the occipital protuberance. Later the vertical incision was introduced, the so-called cross-bow incision of Cushing, and this for a while became the vogue. As time went on we became more and more convinced that the

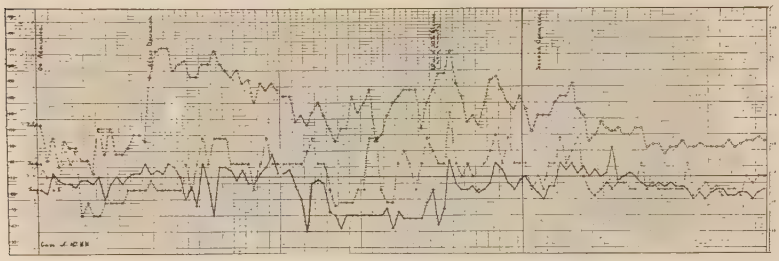

Fig. 5.—Three-stage operation. First stage, January 30, 1926. Suboccipital decompression, followed by great irregularity of pulse and respiratory rate with some improvement after intravenous glucose. (Local anesthesia.)

Second stage, February 11, 1926. Removal of laminæ of second cervical vertebra with extension of the dural incision to that level had immediate effect upon medullary centers. After this second stage the patient's condition improved so that she became ambulant, and her preparation for the last step, March 29, 1926, exposure and removal of the tumor, was unhampered. Recovery.

operation might be very much simplified by eliminating in large measure the horizontal curve and confining the major dissection to the vertical cut with certain additions, about to be described.

By way of preface and before describing the incision in detail, a few words would not be amiss as to the anatomic and other considerations. In the first place, to insure perfect wound repair the major source of blood supply, the arteria occipitalis major, must not be sacrificed. The course of the artery (Fig. 8) may be recalled. It approaches the occipital territory just below the level of the mastoid process, and from this point it passes obliquely upward and inward, until it penetrates the muscle layer and

reaches a more superficial plane. The course of the artery must be clearly visualized by the operator, and under no circumstances should it be cut.

It is essential in this operation that free access be had to the region of the foramen magnum and to the spinous processes of the first and second cervical vertebræ. Hence, the vertical incision, which may extend as low as the fourth or fifth cervical vertebra, is an essential feature of the exposure, relatively, therefore, much more important than the horizontal limb. The longer

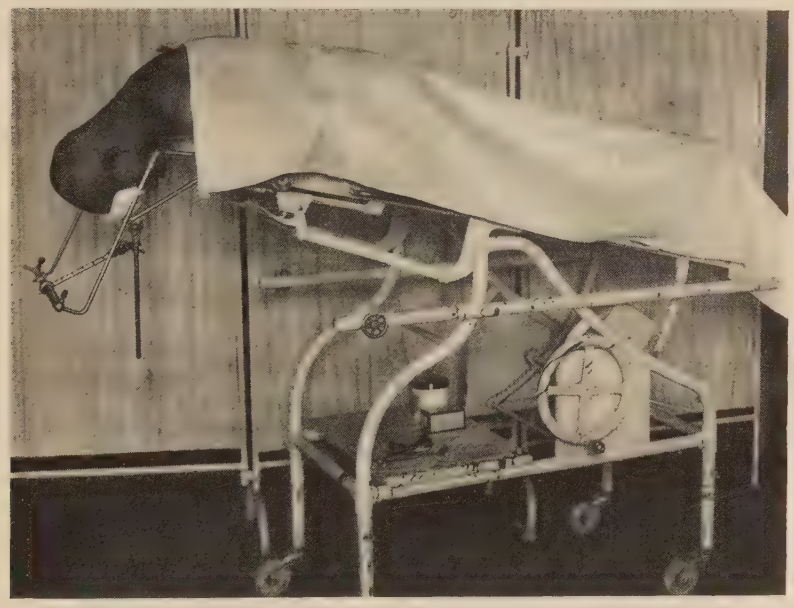

FIG. 6.—Special position for cerebral explorations. Side view.

the vertical limb, the easier the margins of the incision may be retracted.

Many tumors, especially in children, are within the hemispheres, so that without the slightest difficulty the wound margins may be retracted sufficiently to give adequate exposure to either hemisphere after the occipital bone has been rongeured away. If at any time a wider exposure seems desirable on the side of the tumor, a cross-cut may be made in the aponeurosis and muscles on that side, but always below the level of the mastoid process to avoid injury to the occipital artery

Now an objection may be offered to this vertical incision because it does not give an approach to the cerebellopontile angle, a favorite site for posterior fossa tumors. This is perfectly true if it is purposed to approach the angle following the surface of the

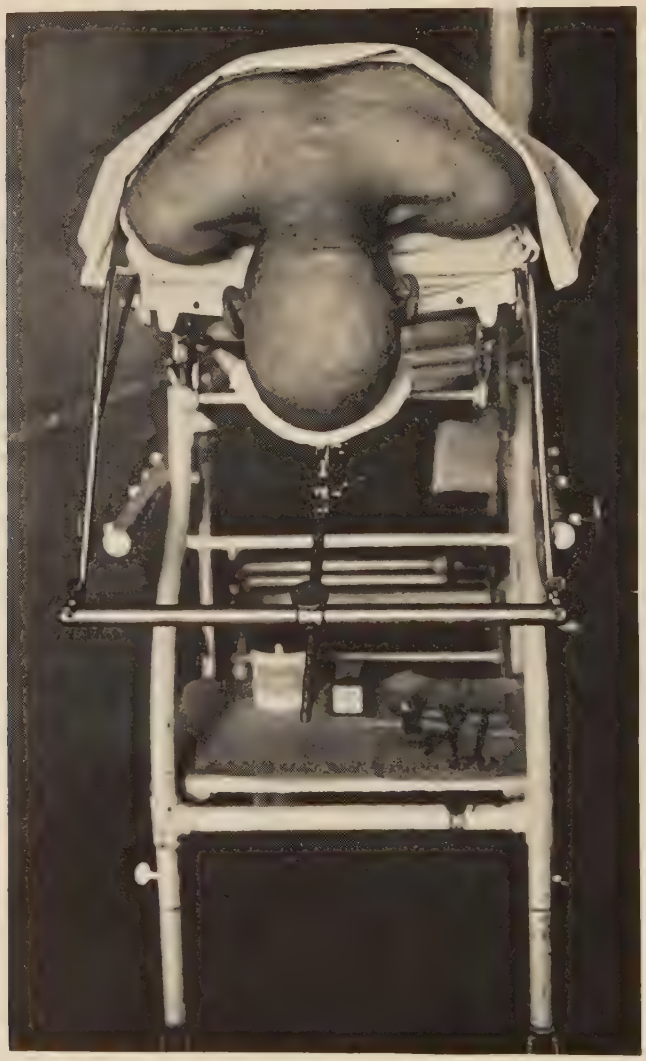

Fig. 7.—Special position for cerebellar explorations. End view.

petrous portion of the temporal bone by displacing the hemisphere toward the midline. However, from a glance at the inner surface of the skull in the posterior fossa it will be seen that the distance to the angle *via* the lateral approach is greater than that from the posterior rim of the foramen magnum to the angle. The most direct and the shortest route to the angle, therefore, is

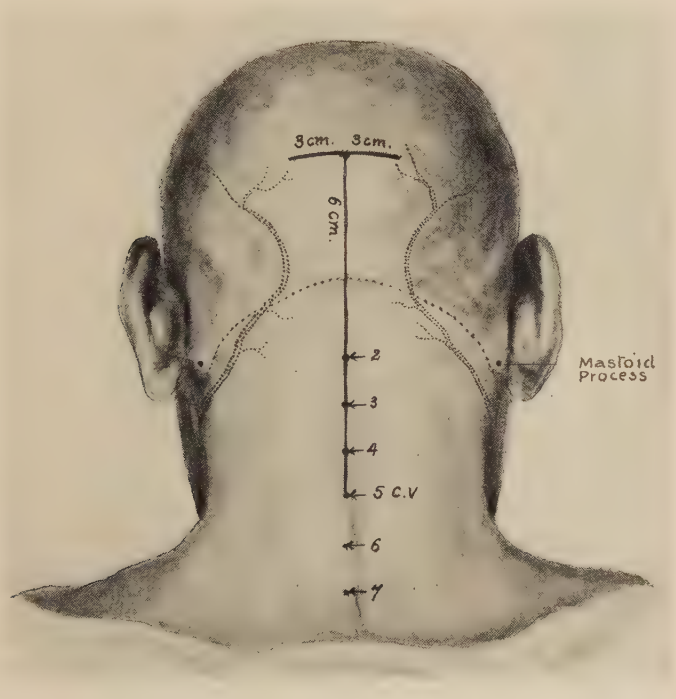

FIG. 8.—Line of incision with relation to (*a*) spinous processes of first seven cervical vertebræ; (*b*) the superior curved line, and (*c*) the course of the occipital artery.

obtained after elevating the cerebellar hemisphere by the mesial approach. If this be true, a wide retraction or reflection of the wound margins or flaps is not requisite and the horizontal limb of the incision may be dispensed with.

An advantage of the vertical *versus* the cross-bow incision is the bloodless field. If one follows the line of cleavage between the right and left muscle groups, with the exception of the vessels

which cross the incision, there is not a vessel below the aponeurosis which requires clamping. As the muscles are separated from the surface of the occipital bone with the pericranium, there is no bleeding even from this step of the operation.

FIG. 9.—Midline incision. Note short cross-cut to facilitate ventricular puncture.

In addition to the muscle cross-cut on one side or the other, it is desirable to make a 3-cm. incision in the scalp on either side of the median line at the upper extremity of the vertical cut, merely as a convenient approach for ventricular puncture (Fig. 9). The latter is so essential a step in cerebellar explorations that provision must be made for it in every instance. Our point of approach for ventricular puncture is 2 cm. to the right or left of

the median line and 6 cm. above the occipital protuberance. These lateral incisions in the scalp are made only to bring these points within the field of operation.

Since we have discarded the conventional cross-bow for the vertical incision, the operation has been reduced to one of relative

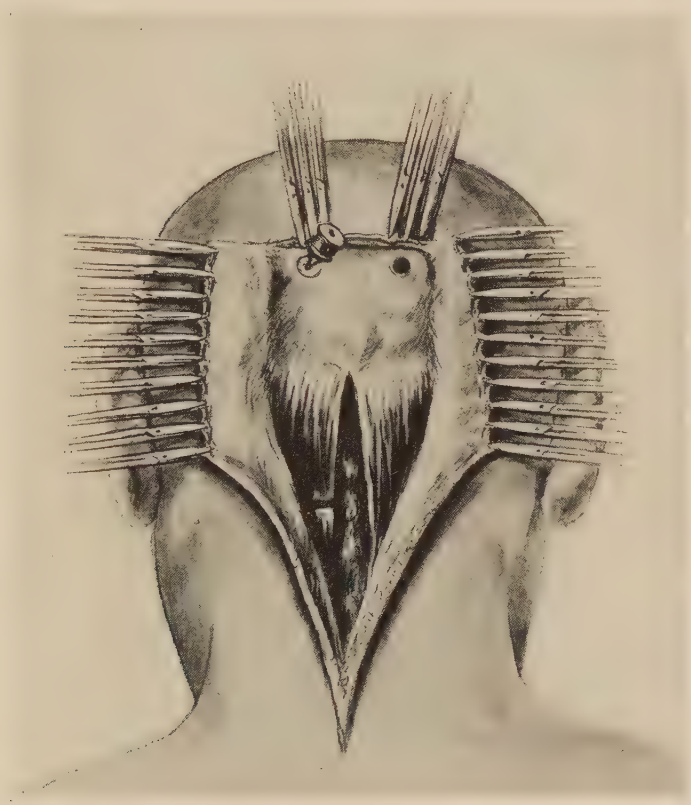

FIG. 10.—Cannula in ventricle on side of tumor. Muscle split in midline of cleavage.

simplicity. The time required to reach the lesion is shortened, a matter of some importance, the approach is through a bloodless field and the time spent in closure of the wound is measurably shortened. With these technical advantages, nothing has been sacrificed to handicap the operator in either the exposure or the removal of the tumor.

Conclusions. The steps of the operation may be briefly described:

1. With the patient in the prone position, head acutely flexed, the line of the projected incision is infiltrated with 0.5 per cent novocaine solution from a point 6 cm. above the occipital pro-

Fig. 11.—Bisection of muscle mass at low level avoids cutting occipital artery and gives adequate exposure to the side of the lesion.

tuberance to the level of the spinous process of the fifth cervical vertebra.

2. The superficial incision is made along the line of infiltration down to the muscle plane; the hemostats are applied to the deep fascia before digital pressure is released and the wound draperies sutured in place.

3. After infiltration of the deeper plane of tissues the deep incision is continued following precisely the bloodless line between the muscle groups of either side and down to the cranium and the tips of the spinous processes. To avoid pain the pericranium is desensitized with the novocaine solution and the muscles, with

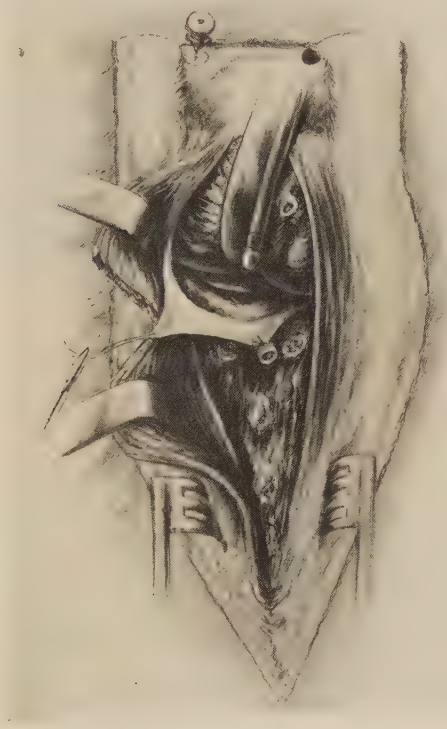

FIG. 12.—Slight elevation of the hemispheres shows the tumor from the angle presenting in inferior surface of cerebellar hemisphere.

the pericranium, separated on either side from their attachment to the occipital bone. If later a wider exposure is required, the muscle layer only on the side of the lesion is bisected below or at the level of the mastoid process.

4. After perforation of the skull the bone is removed on either side, as in all suboccipital exposures, from below the occipital

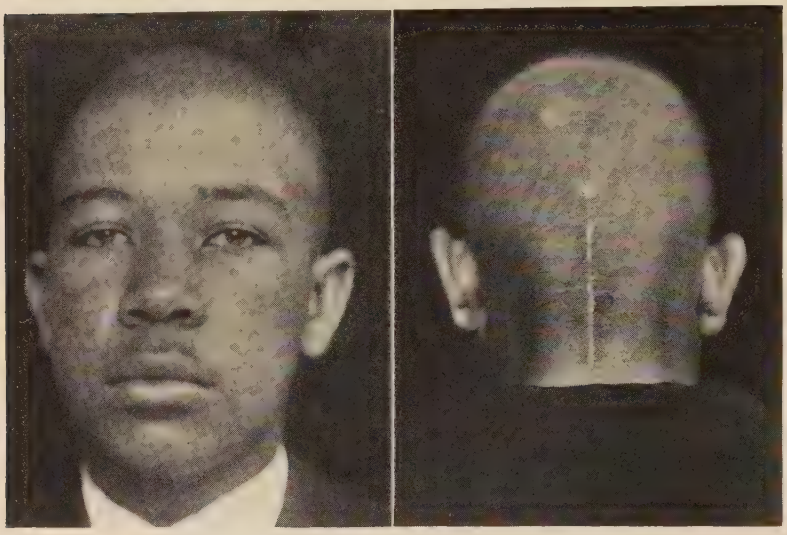

FIG. 13.—Photograph of patient from whom a cerebellopontile angle tumor was removed.

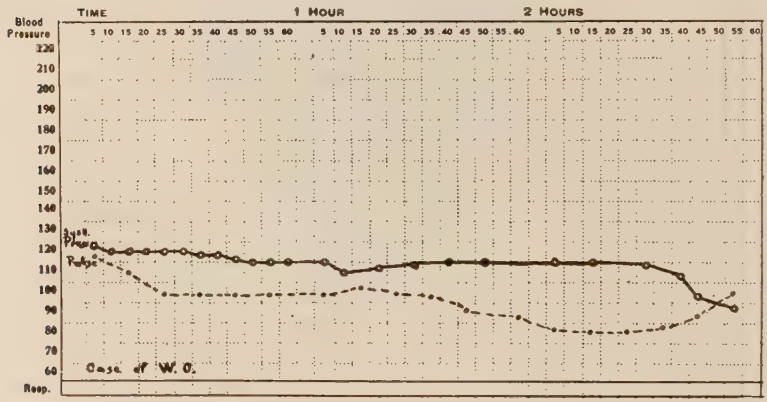

FIG. 13*a*.—By observing these safety factors, local anesthesia and the two-stage operation, if necessary, the hazard of the cerebellar tumor operation has been measurably reduced. Note uniformity of pulse and blood-pressure during exposure and removal of vascular sarcoma.

protuberance to and including the posterior rim of the foramen magnum together with spinous process and lamina of the first cervical vertebra. The bony defect should extend out to, but not beyond, the emissary vein on either side.

5. Before the dura is reflected the occipital sinus must be clamped with mosquito forceps, divided and a ligature applied to distal and peripheral segments. When dividing the dura, care should be exercised to avoid cutting or lacerating the arachnoid membranes.

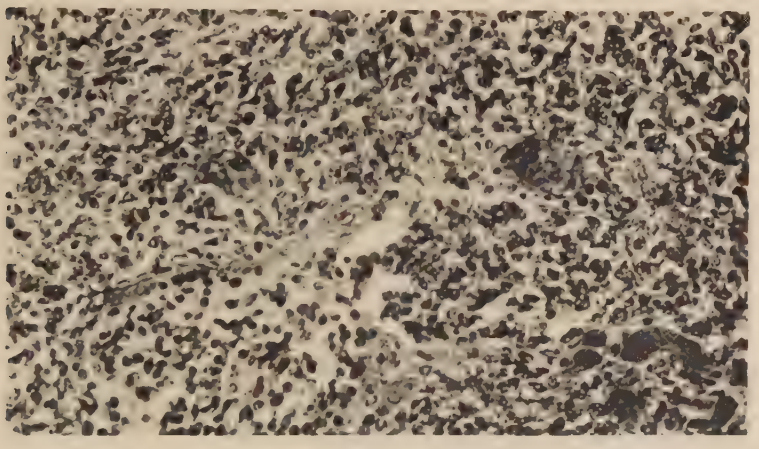

FIG. 13*b*.—Pathological slide; high-power photomicrograph; sarcoma.

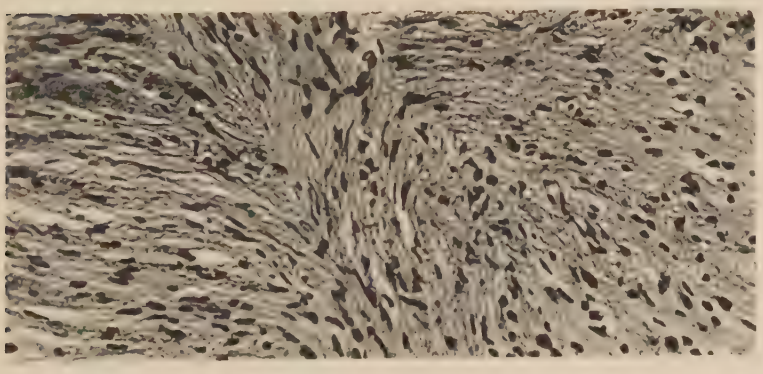

FIG. 14.—Pathological slide; high-power photomicrograph; endothelioma.

This concludes the essential features in the suboccipital exposure as peculiar to the method advocated. The subsequent steps of the operation naturally vary according to the location of the lesion. If the tumor be within the hemisphere it is readily accessible; if it be situated in the cerebellopontile angle the hemisphere is displaced upward.

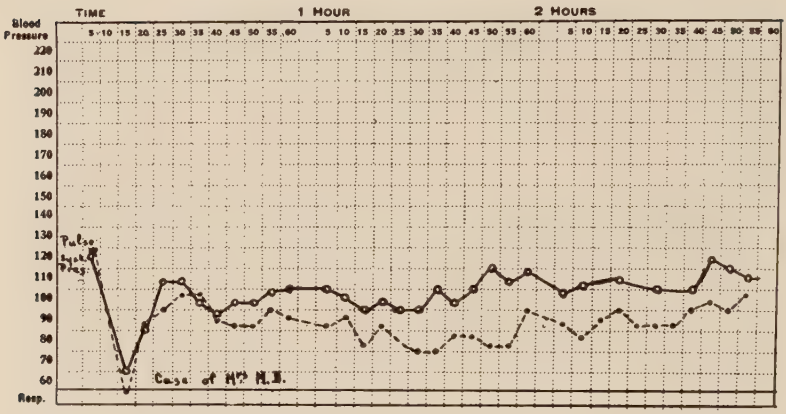

FIG. 14*a*.—Blood-pressure chart. Note freedom from disturbance of blood pressure or acceleration of pulse throughout the operation. (Local anesthesia.)

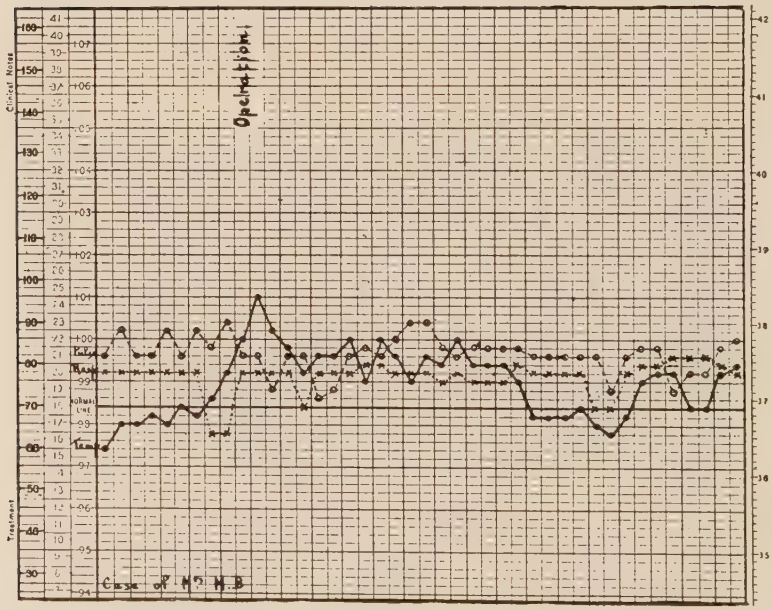

FIG. 14*b*.—Temperature chart. Note slight disturbance in pulse, respiration-rate and temperature reactions following removal of angle tumor.

DISCUSSION

Dr. W. J. Mixter, Boston, Mass.:

The midline approach, as suggested by Dr. Frazier, has been somewhat difficult in my hands; but having watched Dr. Frazier do it once or twice, and having seen his presentation today, I feel it is worth a great deal in certain types of cases.

CHOLESTEATOMATOUS CYSTS: WITH REPORT OF CASE INVOLVING THE MAXILLA

By JOHN M. T. FINNEY, M.D.

AND

EDWARD M. HANRAHAN, Jr., M.D. (By Invitation)

BALTIMORE, MD.

Although cholesteatomatous cysts occur not infrequently in various parts of the body, we have recently observed one whose unusual location merits recording.

Our patient was a white woman, aged sixty-four years, who complained of a swelling of the left side of her face. Her history was irrelevant except for the fact that all of her teeth had been extracted ten years previously. She states that this was done because of a marked grade of pyorrhea alveolaris. Four months before our examination it had been noted that the left side of her face appeared swollen. She consulted a surgeon and a roentgen-ray examination was made. No treatment was given, until her family physician, noting a progressive increase in size, sent her to Baltimore for an examination. At no time has she had pain or inconvenience as a result of this swelling.

Her physical examination was negative except for the condition noted. The left side of her face was the seat of a swelling located just beneath the zygomatic arch. It appeared to extend below to the maxilla and upper alveolar border. The skin of the cheek was normal in appearance. The left lid-slit was slightly narrower than the right. On palpation there was felt a bony-hard, smooth rounded tumor about the size of an English walnut and measuring 4 cm. in diameter. All of the teeth had been extracted and the gingival mucous membrane was in good condition, although dental plates had been worn. The swelling extended from about the site of the upper lateral incisor to approximately the location of the second molar. It obliterated the outline of the alveolar process and was covered with normal appearing mucous membrane. It was hard and fixed, neither tender nor painful and

toward the posterior border a distinct egg-shell or ping-pong ball crepitus could be felt. The lymph glands beneath the angle of the left jaw were definitely larger than on the right side.

FIG. 1.—Roentgenograph of cyst occupying the alveolar border of the maxilla.

A roentgen-ray examination was made and interpreted by Dr. F. H. Baetjer as follows: "Arising from the hard palate and projecting anteriorly there is a tumorlike growth. This growth is completely surrounded by a bony shell except at its anterior portion. This rather

gives one the impression that we are dealing with a partly encapsulated tumor, semibenign. In some respects it is more like a giant-cell than a round-cell sarcoma."

At operation, in May, 1922, an incision was made over the obliterated dental line. A thin shell of bone was encountered which was easily pierced with slight pressure of the knife. Directly on opening into the cyst cavity there was a discharge of brownish-red, viscid contents. It was noted that this material contained particles which reflected light almost like mica. The cholesteatomatous character of the cyst was immediately recognized and it was thought that the condition could best be treated by a thorough removal of the lining and the greater part of the thin bony wall. This was done and the remaining cavity packed with iodoform gauze. There was no connection found between this cyst and the immediately adjacent antrum. Healing by granulation and epithelialization took place uneventfully.

The contents of the cyst were carefully examined; they were of uniform consistency and were composed of inspissated material—yellowish pigment and quantities of mica-like particles. A smear of this material when seen under a microscope showed much cellular debris and typical cholesterine crystals, measuring about 1 to $1\frac{1}{2}$ mm. in length. There was found neither hair nor any solid particles.

A section of the cyst wall showed thin and flattened squamous epithelium separated from the bony shell by a very thin layer of connective tissue. This thin layer contained partially or completely atrophied dermal glands and is to be considered as representing corium. The cyst wall, therefore, exhibits the typical structure of a dermoid cyst.

The patient was examined December 3, 1925 by Dr. Frank C. Wilson of Birmingham, Ala., who kindly reported that he had found a cavity in the upper jaw extending to the midline. This cavity was lined with normal mucous membrane. There was no evidence of any tumor.

The possibilities regarding the origin of this cyst interested us greatly. Whether its epithelial nature has to do with dental structures or with one of the simpler forms of epithelial cysts, we cannot be certain. The solution of the question would, perhaps, be more definite were it not for the history of pyorrhea alveolaris preceding the extraction of the teeth. This brings up the possibility of an inflammatory origin, which must be considered.

The various cysts and tumors of the maxilla are believed in most cases to have a dental origin. Malassez, in 1885, referred most of these tumors to embryonal remnants of the enamel organ. He

found in the fetus many small groups of cells scattered along the roots of the teeth, which he designated "paradental epithelial débris." It is Ewing's opinion that these cell groups are derived from the invaginations of gingival epithelium which go to make up the enamel organs, and that dilatation of these vestigial structures gives rise to the various simple cysts found in contact with or in the neighborhood of the roots. The single or multiple cysts of the maxilla, containing walls or cavities and imperfect or well-formed teeth, appear in many cases to be derived from these paradental structures and from the specialized mesodermal element over which the enamel organ exerts a formative control. The sole source of the entire group of cystic and solid malignant epithelial growths arising in the maxilla is, according to Ewing, to be found in the paradental epithelium. The extent to which the dental follicle itself is concerned in the formation of these dentiferous and dentigerous cysts is uncertain. It is believed that the more complex growths arise from the original or from supernumerary dental follicles. The transitions which may occur between simple groups of paradental epithelium and true supernumerary enamel organs will account in some measure for the variation in structure and contents of maxillary cysts.

These cysts and tumors of the maxilla may be divided into two groups, depending on their relation to the teeth: Adamantinoma and radiculodental cysts are considered to be related to erupted normal or carious teeth. Coronodental and dentigerous cysts affect the tooth before its eruption, and may prevent its full development.

Adamantinoma arises from the paradental epithelial débris. It may be solid or cystic or both. When solid, it may be very cellular and malignant. The cells vary from simple squamous epithelium to the more specialized enameloblasts. In the solid form its appearance sometimes suggests a round- or spindle-cell sarcoma, but the peripheral arrangement usually indicates its true character.

The cystic form may be single or multiple, or there may be a papillary form containing small cysts. The contents of the cyst may be serous, mucoid, inspissated fatty or caseous material. Adamantinoma usually occurs in the adult, and there may be a history of extraction of a carious tooth.

Radiculodental cysts are attached to the roots of normal or

carious teeth and are thought to arise from the deep paradental epithelial débris by proliferation and dilatation of these structures. The smaller cysts may suppurate and, the epithelial lining being

FIG. 2.—Bony shell on left, with cyst wall on right. The inner lining of the cyst wall consists of a thin layer of stratified squamous-cell epithelium. The tissue between this lining and the bony shell represents corium.

destroyed, appear as a small abscess. Several large cysts apparently of this origin have been described. Bland-Sutton encountered several, and his examinations confirmed Turner's observation that they are lined by stratified epithelium and also that the

contents often contained cholesterine crystals. This structure has suggested a dermoid origin. Larger cysts have also been described by Sirantoine, and Witzel believes that they arise from dilatation of von Brunn's sheath. This sheath is a continuation of enamel epithelium over the root of the tooth, and is included by Malassez with the paradental débris.

Coronodental cysts, dentigerous cysts and odontoma usually occur in young adult life, before the full development of the teeth has been reached. Coronodental cysts are usually small cavities, whose walls are lined with epithelium and into which project one or more deformed teeth. Dentigerous cysts and odontomas include those cysts or solid tumors caused by an increase in number of more or less well-formed teeth. The cysts occur in various forms. There may be cysts containing one or more teeth or the teeth may be multiple and usually deformed. The odontoma is made up of aggregates of these teeth, sometimes with cysts. Localized growths of dental structure are related to odontoma. The different forms doubtless present different modes of origin. When the normal tooth is absent, its follicle is probably the source of origin of the tumor. When normal teeth are present, abnormalities may be traced to supernumerary follicles, or the less orderly growths to the paradental epithelial débris.

This brief résumé of the cysts and tumors of dental origin is possibly necessary in order properly to evaluate the suggestion that the cyst in our case may be of dermoid origin. Epithelial cysts are usually atheromatous, implantation or traumatic dermoids, congenital epidermoids or true fissural dermoids. It is to the latter variety that this cyst may be related.

The true fissural congenital dermoid is located in those regions of the body which may be considered subject to embryonal disturbances of development. Their common occurrence in the scalp, neck, back, along the median line of the chest and abdomen, sacral region and buttocks is in accord with their relation to embryonal fissures, clefts and junctures. The cyst wall may present all the structures of the skin, including epidermis, dermis and dermal glands.

Of the more common dermoids, the intracranial variety is interesting in this connection, owing to the occurrence of the intracranial cholesteatomata. Cushing, Horrax and Bailey have fully discussed these unusual tumors, which were called by Cru-

veilhier "tumeurs perlées," and by Bostroem, "piale epidermoide." According to Ewing, the relation of the medullary groove to the ectoderm, the complex steps in the formation of the brain and ventricles, and the formation and union of the cranial bones give

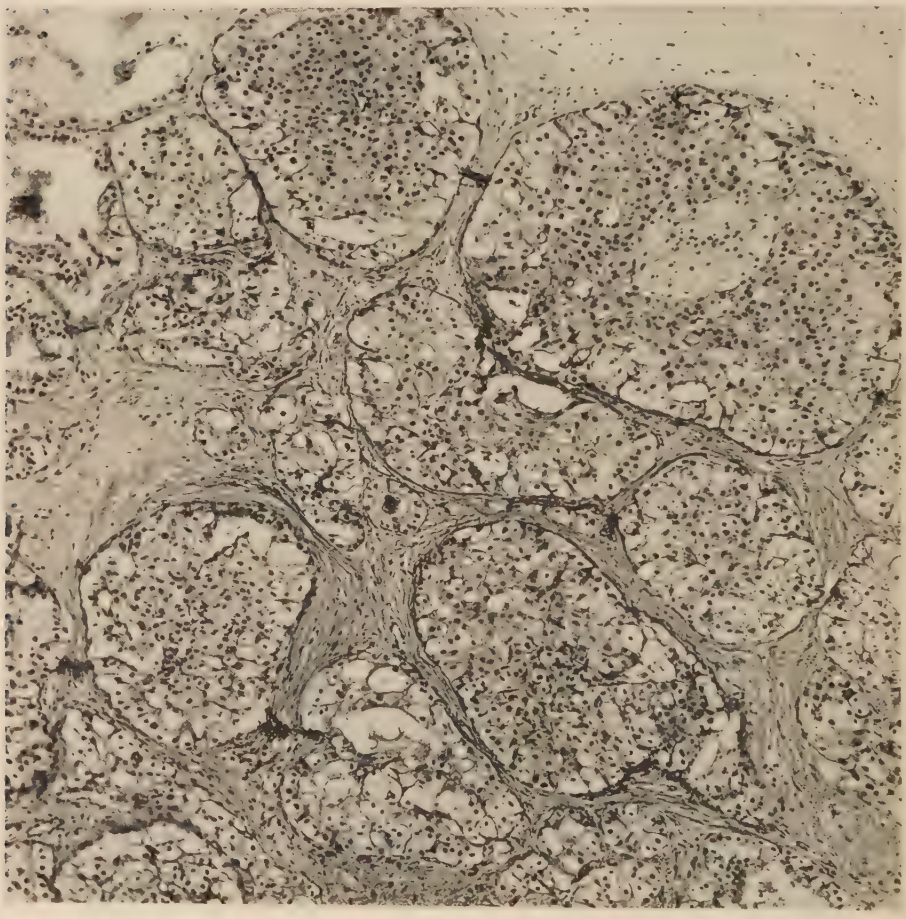

FIG. 3.—Atrophic mucous (dermal) glandular tissue found in corium of cyst wall.

abundant sources for the development of epidermal growths within the skull. The interpretation of these tumors is further complicated by the occasional tendency of endothelial growths to copy the structure of cholesteatoma, and finally, traumatic implanta-

tions of portions of ectoderm account for a small proportion of intracranial processes. Bailey found that the favorite location for these collections of epithelial cells is in the pia mater around

FIG. 4.—Contents of cyst, showing the crystaloid character. Giant cells are also to be seen. A smear of this material immediately after operation showed quantities of typical cholesterine crystals.

the base of the brain. They may project upward into the cerebellopontine angle, the fissure of Sylvius, between the frontal lobes, or into the third ventricle. They occur relatively often extradurally, under the parietal or temporal bones. Rarely, they

may be found in the fourth ventricle or, more frequently, in the middle-ear cavity. Bailey felt that no better example could be found of a tumor originating in a group of epithelial cells separated from its normal surroundings and developing autonomously.

One mechanism of this inclusion of epithelial cells is the faulty separation of skin and dura when the membranous cranium chondrifies. The resulting epidermal rest may be outside or inside the bone, where it would project on the inner surface or between layers of dura.

The histologic description of Bailey of his pearly tumors is much the same as that of our maxillary cholesteatomatous cyst. The pearly tumors are composed of a fine fibrous wall whose inner surface is covered by layers of flattened cells containing keratohyaline granules, and also masses of cells flattened and structureless or with only the intercellular substance remaining, giving the appearance of the cells of woody plants. The interior may be a broken-down mass of débris containing fatty material and cholesterine crystals.

While the majority of cysts and cystic tumors of the maxilla undoubtedly have a dental origin, it is quite possible that epithelial cysts in this region may arise from a defect in the fusion of the premaxillary process with the maxilla proper. It will be remembered that the premaxilla has, according to Albrecht and Warinski, two centers of ossification. These centers appear about the eighth week, and by the tenth week have fused together so that the maxilla consists of two portions, the premaxilla and maxilla proper. The suture between these two sometimes persists into adult life, and may be seen as a delicate line which extends forward and laterally from the anterior palatine fossa to the interval between the lateral incisor and the canine tooth. This would correspond to the anterior border of the cyst observed in our case. It is quite possible that the inclusion of an epidermal rest at this point of fusion would furnish a genesis quite apart from any dentigerous origin, which would be entirely compatible with the fissural origin of a true dermoid.

The occurrence of a cystic tumor of the histologic structure described, and its origin in this particular location, affords such interesting speculation as regards embryology and pathology, as to justify our taking up the time of the Association with this brief communication.

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THE TREATMENT OF CUTANEOUS BURNS

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NEW YORK CITY

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The disappearance of the gas and kerosene lamps has not diminished the number of burns brought for treatment to metropolitan hospitals to the degree that one might expect. Lighted Christmas trees, gas-stoves, exploding stills, boiling water, automobile and industrial injuries produce still an astounding number of burns. The toll from these injuries remains depressingly high—deaths, immediate and delayed, are too frequent.

All surgeons must eventually appreciate the economic loss through prolonged hospital stay. This loss is borne not only by the patient but also by the public, for rarely does a ward patient pay his per capita expense to the hospital. Our figures show an instance of 36 per cent of burns in children under ten years of age. Deformities occurring at this age may result in their being public charges for many years. The memory of a burned child remains with the surgeon not only during the day, but often if he chances to lie awake at night it may haunt him, particularly if he has a guilty feeling that at some time in the course of the treatment he might have done more to improve the child's condition. We feel that much must be done by public education both of mother and child to prevent this appalling percentage of burns in children.

As one reviews the literature, one sees that the pendulum of interest in these deplorable accidents of civilization swings as it does elsewhere in medicine. At times surgeons are stimulated to extreme interest and this is reflected in the literature. Too often there is a lack of interest and these unfortunate patients are delegated to the most inexperienced house officer to dress and

care for. No patient needs more painstaking care, day in and day out, unfortunately even month in and month out. Their treatment demands a knowledge of blood chemistry and clear, sane surgical judgment. Dr. John Staige Davis has said that each extensively burned child needs to have not one but twenty varieties of treatment, of operative procedures, of forms of skin grafts, and that no one rule can fit all burns—no, not even a single large burn. But if a single rule should be followed, the only one is the use of extreme patience and constant reorganization of therapy.

Our purpose in presenting this paper is to offer nothing new, but to review the work of others -present some of the problems and pitfalls that have occurred during the treatment of a fairly large number of burns that have been admitted to the Lincoln Hospital during the past two and a half years. We, at first, used the débridement method and lately have used tannic acid. We have had opportunity to observe results from both methods and desire to offer our conclusions.

Burns are usually described as first, second and third degree. We believe that from a therapeutic point of view the present third-degree burns should be divided into third and fourth degree.

Third-degree burns are those that destroy the epithelium but do not destroy the hair follicles.

Fourth-degree burns are those wherein all the epithelium and the subtaneous fat is necrosed.

It is obvious in this classification that, during repair, islands of epithelium might arise from the epithelium of hair follicles in third-degree burns which would be absent in fourth-degree burns. The importance of this differentiation will be brought out in the discussion of débridement of the necrosed tissue.

Death due to burns may be classed under two headings: (1) Shock; (2) Toxemia.

1. In this group are cases dying within twenty-four to thirty-six hours with symptoms of so-called shock, as evidenced by rapid pulse of low volume, coma or only partial consciousness, and subnormal temperature with a cold, clammy skin.

On admission morphine is given immediately. External heat in the form of hot-water bottles is applied wherever possible. In other cases the electric cradle is used. The administration of fluid has its part in the treatment of shock. Hypodermoclyses are given at repeated intervals and the Murphy drip is also made

use of. We believe that one of the most important measures that should be used is early blood transfusion. This is to be done as near to the time of injury as possible, since its value is inversely proportional to the length of time that elapses between the injury and its administration. This last measure will often balance the scale in favor of a border-line case.

Where it is impossible to obtain a donor for transfusion, or the facilities are not adequate, glucose intravenously is of great assistance.

2. Numerous theories have been advanced to account for the deaths of patients dying from eight days to three weeks with symptoms suggesting toxemia. Early writers thought this was due to interference with the skin functions and offered as examples the death of gilded experimental animals, but later evidence showed that these deaths were due to metallic poisoning. Robertson and Boyd burned the skin of young rabbits with hot metal plates. In one series of experiments the burned skin was removed within eight hours and grafted on unburned rabbits, while the skin of the unburned rabbits was grafted to fill the defect of the burned. Their result showed development of toxemia in the rabbits receiving the burned transplants, while the others escaped. If the transplants were made after eight hours, both burned and unburned suffered. Robertson and Boyd further showed that the serum of burned children when injected into rabbits was innocuous. The injection of citrated whole blood caused typical toxemia. Their conclusions were as follows: Toxic substances consist of two portions—one of which is thermostable, diffusible and neurotoxic; the other is thermolable, colloidal and necrotonic. Chemically the toxin consists of primary and secondary proteoses.

Underhill and his co-workers found a marked concentration of the blood in a study of 21 cases—the hemoglobin in the serious cases varied from 150 to 209 per cent of normal. Underhill concludes as follows: "Marked concentration of blood means a failing circulation, an inefficient oxygen carrier, oxygen starvation of the tissues, fall of temperature and finally suspension of vital activities." Similar conditions are found in war gases and in certain cases of influenza. He thinks this concentration is due to the constant pouring out of fluids on the burned surfaces. He believes an animal cannot long stand a concentration of 140 per cent of normal value. Where the blood has been concentrated

the sodium chloride content of the blood is low. In other words, the sodium chloride content varies inversely as the hemoglobin.

Davidson, in a recent paper read before the Surgical Section of the New York Academy of Medicine, reported similar findings of sodium chloride depletion and suggested the analogy between the toxemia of intestinal obstruction and burns.

Davidson says: "A third group of 12 patients was investigated in which the burned area varied in extent from 1000 square centimeters to 24 per cent of the total body surface. In these cases the blood chlorides remained at a very low level as long as sloughs were present. Upon separation of the devitalized tissue, when absorption was no longer taking place, the blood chlorides rose, and a parallel rise of the urinary chlorides was noted. The depressed values of the blood chlorides could not be attributed to diet, blood concentration, altered renal threshold, fever, nor to loss of plasma from one-half of the burned surfaces. The evidence suggested that there was retention of sodium chloride in the tissues in general quite similar to that observed in pneumonia."

"In view of these findings it would seem rational to correct this metabolic deficiency by the administration of sodium chloride. Further studies are necessary to determine whether sodium chloride will spare body protein similar to that observed by Haden and Orr in intestinal obstruction."

If we assume that the late deaths in burns are due to a toxemia liberated by the injury, our treatment must be based, first, to combat the toxemia; second, to prevent as far as possible the absorption of the toxin into the blood stream.

1. Treatment of Toxemia. In a great measure, the treatment of toxemia is similar to that of shock, save that it must be continued over a longer period of time. Great emphasis is placed on fluid administration and the elimination of waste products. Again hypodermoclysis and fluids by mouth are forced. Attention should be paid to the local skin condition from which the absorption of large amount of toxins takes place, unless the toxin material is either removed by débridement or taken by other measures.

It is vital that secretion, which is profuse from the granulating surfaces, be removed carefully and painstakingly to prevent severe infection. One very great factor in the prevention of these surface secretions after the sloughs have separated is early skin grafting.

As these patients go on from week to week they develop a more

or less severe secondary anemia. There is a persistent temperature due to slow absorption of toxic products. For them the greatest benefit is to be derived from blood transfusion. Its value cannot be overestimated. This should be done as often as necessary until the patient can carry on alone.

Exsanguine transfusion has been advocated, but it seems to us a very heroic procedure. Accidents might happen during the transfusion necessitating its discontinuance. Furthermore, it can only be of very transient benefit, as toxins are being constantly poured into the blood stream.

The necessity of fluid intake, often by means of hypodermoclysis, is shown in one of our cases. A woman, aged thirty-two years, was admitted April 1, 1926. Her clothes had caught fire while reading by candle-light and she had extensive second- and third-degree burns of abdomen, chest, buttocks and thighs. She was treated by tannic acid for twenty-four hours. Two days after admission she was vomiting persistently. On the second day she was given 3000 cc. by hypodermoclysis and on the third 2000 cc. After the first hypodermoclysis her vomiting ceased. Four days later she again started vomiting but was immediately relieved by hypodermoclysis.

2. Treatment of Local Condition. We at first attempted the débridement method as advised by Brager, Lieber and Willis. Efforts were made to see if we could determine on admission the line of demarcation between the second- and third-degree burns. This proved very difficult, so we waited until about the third day, when the patient was taken to the operating room, anesthetized, and the area cleansed thoroughly. The skin and fat were then débrided, frequently to the fascia. The fat seemed to us to be very severely damaged. It did not bleed actively and had a peculiar yellow-gray appearance. Whenever possible and necessary, the patient was transfused at the same time. Very frequently marked general improvement was noted in a few hours—as evidenced by improvement in the pulse, temperature and mental attitude. The wound was treated postoperatively with vaseline gauze.

The following 2 cases were cited as showing the marked immediate improvement following débridement.

Case I.—V. K., a girl, aged twelve years, admitted January 1, 1925, with severe second- and third-degree hot-water burns over the buttocks,

thighs, legs, feet and left hand. On her sixth day she was intensely toxic and was so ill that we doubted if she would live twenty-four hours. Under light anesthesia the typical pig-skin appearing burnt area down to fascia was completely removed from the right thigh and leg—a similar procedure was attempted on the left leg but her condition was so serious that in many places the area was rapidly incised to the fascia by gridiron-like incisions. This was followed by a transfusion of 250 cc. The next day her condition was markedly improved, and signs of toxemia gradually disappeared. She progressed to a complete recovery.

Case II.—E. B., aged fifty years, admitted October, 1925. A very obese woman had extensive deep burns of back from the neck and dorsal surface of her arms to below the scapulæ—she was semistuporous on admission. The following day she was delirious, with a rapid pulse and temperature of 103° F. She looked as if death were imminent. A débridement of an area 8 x 10 inches was rapidly performed. The skin resembled parchment. She was immediately transfused with 450 cc. whole blood. The improvement was startling. On the following day she was conscious, pulse was about 90 and of good quality. Her condition remained excellent until about the twelfth day, when her temperature became elevated and she died on her twenty-first day with symptoms suggesting infection, although the wound appeared relatively clean.

We believe that both of these cases would have died immediately had not the toxic tissues been removed or the lymphatics and capillaries blocked by some chemical agent such as tannic acid. The last case was a very poor risk, a patient with hypertension and damaged kidneys; she was unable to survive the large tissue damage.

The disadvantages of débridement are obvious. 1. It is a radical procedure with considerable operative risk to a devitalized patient. Islands of epithelium in the hair follicles, sweat and sebacious glands may be sacrificed that might be saved.

2. The after-treatment is extremely painful and infection is very apt to follow.

3. Skin grafting must usually be performed to cover the defect. If the condition of the patient is poor and infection present, skin grafting may be so long delayed that a scar-tissue base may form beneath the granulation tissue and a second extensive débridement must be performed to make a satisfactory field for the grafts.

Pathology. Sections are presented of 2 cases where débridement was performed. Case I, two days after injury. Case II, six days after injury.

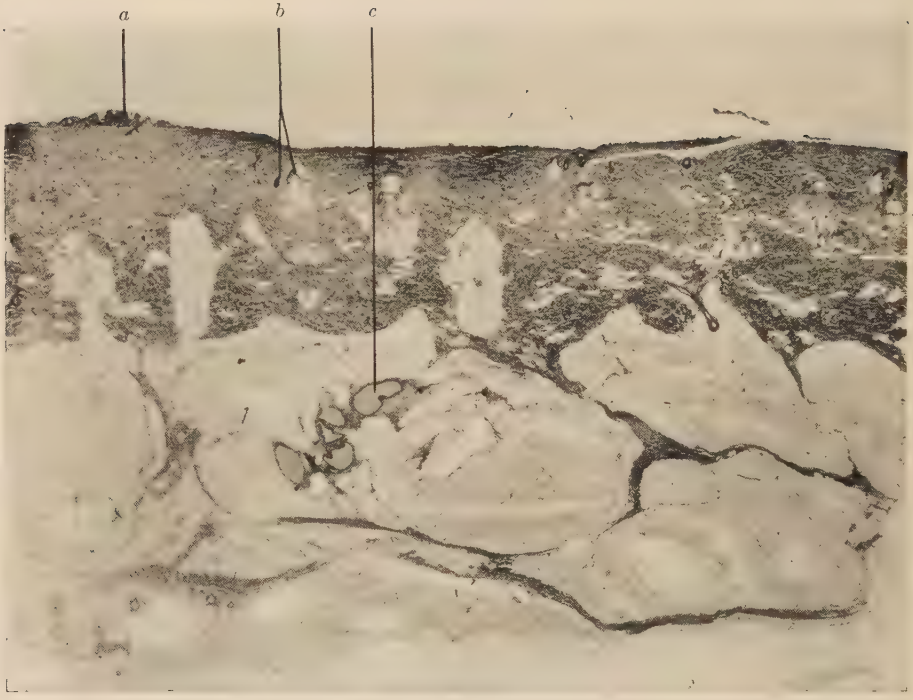

FIG. 1.—Case I. Low-powered view débrided subcutaneous tissue and fat, two days after burn. *a*, Remnant of epithelium. *b*, Hair follicle and sebaceous glands. *c*, Bloodvessels.

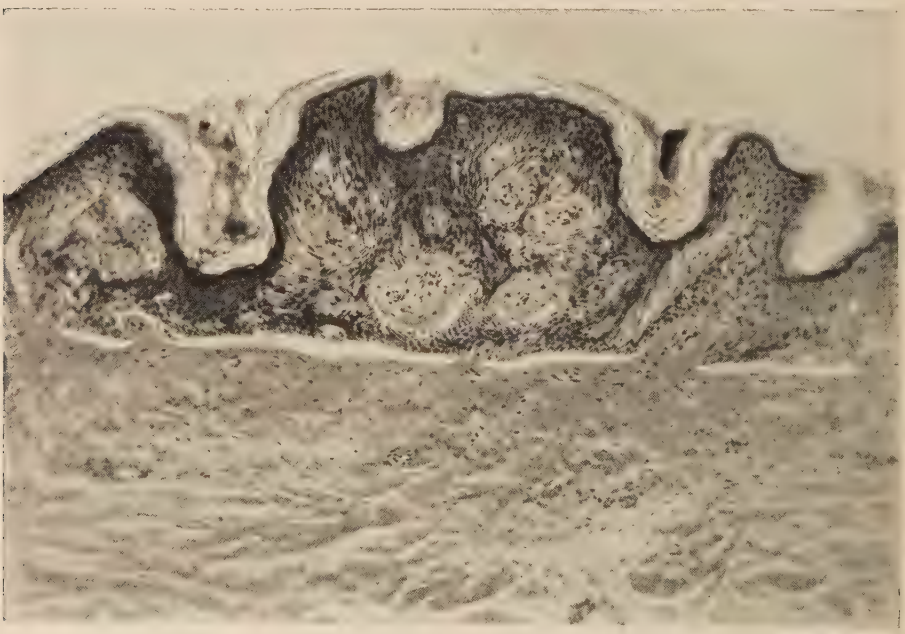

FIG. 2.—Case I. Remnant of epithelium showing epithelial pearls, injury to epithelium, edema and granular degeneration of connective tissue.

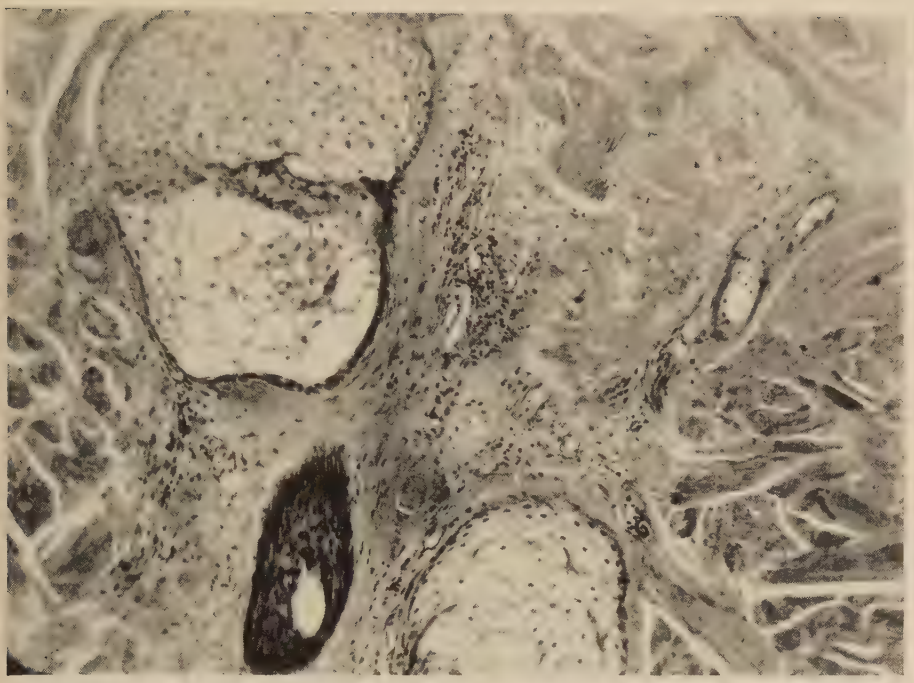

FIG. 3.—Case I. Hair follicle and sebaceous gland. Hair follicle with fairly normal appearing epithelium. Granular degeneration of connective tissue, polymorphonuclear and mononuclear infiltration.

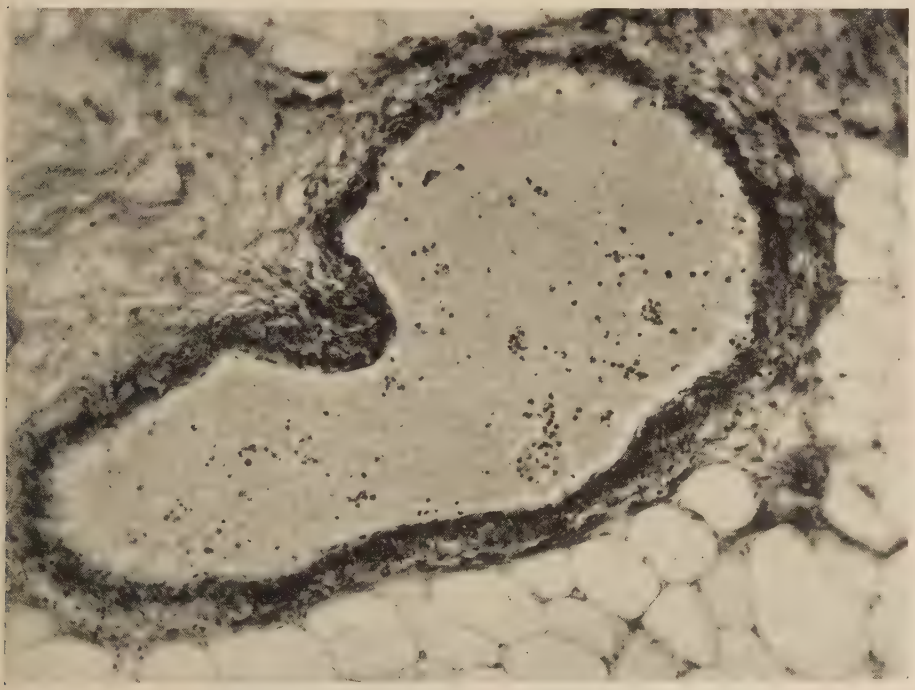

FIG. 4.—Case I. Bloodvessels with walls markedly traumatized and apparently coagulative necrosis of the blood.

FIG. 5.—Case I. Shows an epithelial destruction of sweat glands and a granular degeneration of connective tissue.

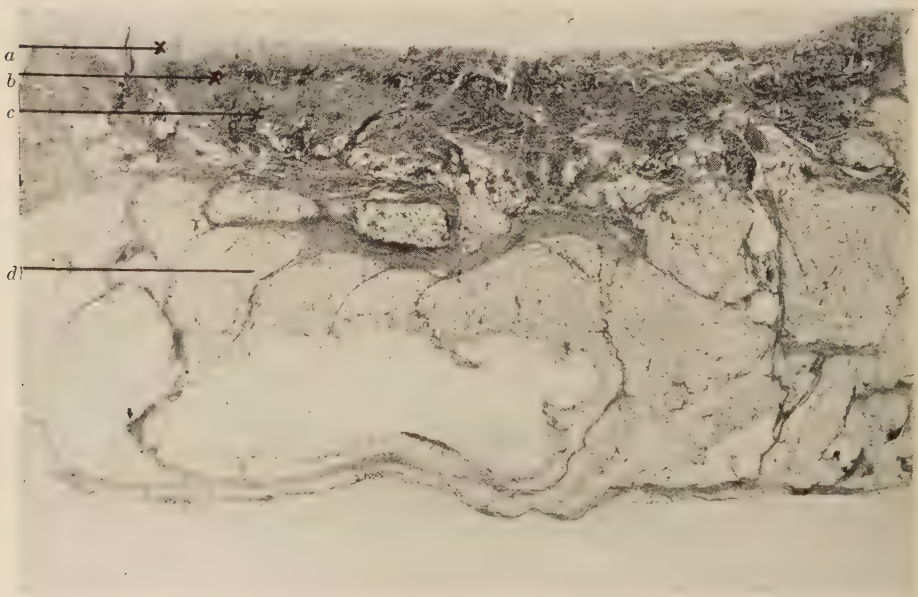

FIG. 6.—Case II. Low-powered view, débridement six days after burn. *a*, Necrotic tissue. *b*, Separation zone. *c*, Living connective tissue. *d*, Fat.

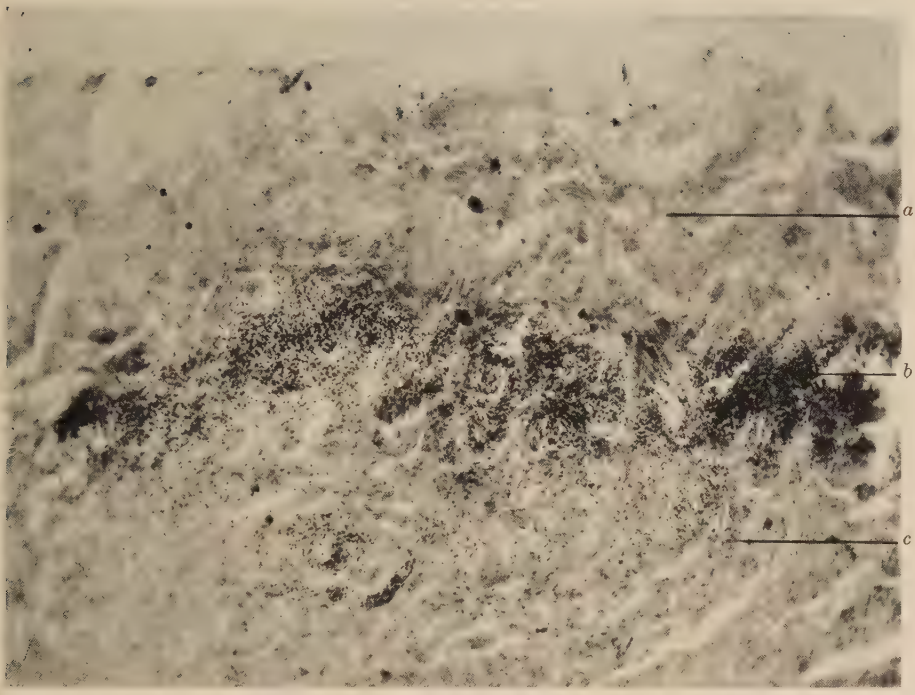

FIG. 7.—Case II. High-powered view of separation zone. *a*, Necrosed tissue. *b*, Separation zone. *c*, Connective tissue showing presence of infection.

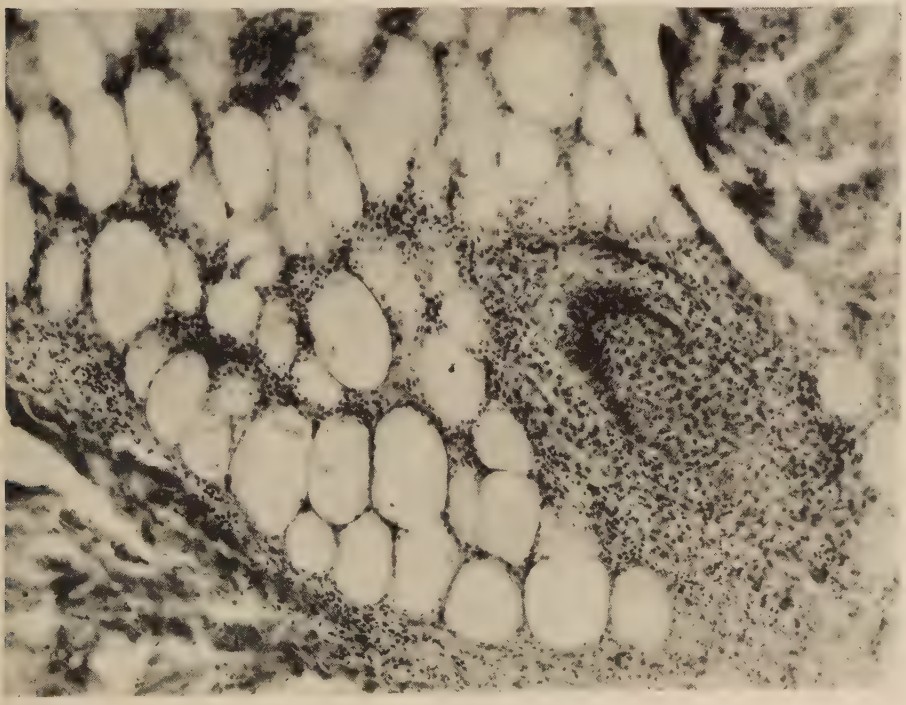

FIG. 8.—Case II. Shows an infection of fat and hair follicles.

FIG. 9.—Case II. Marked cellulitis of fat and subcutaneous tissue and infection about sweat glands.

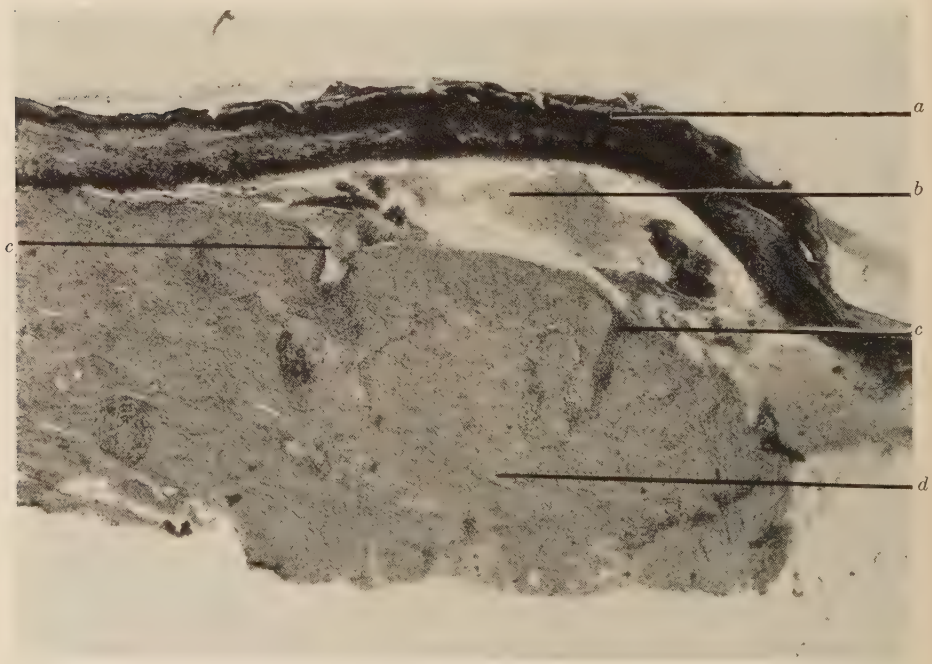

FIG. 10.—Case III. Low-powered view, child aged eight years, treated by tannic acid. Section removed four days after burn. *a*, Tannic acid membrane. *b*, Separation zone. *c*, Hair follicle. *d*, Subcutaneous connective tissue. (See Figs. 11, 12 and 13.)

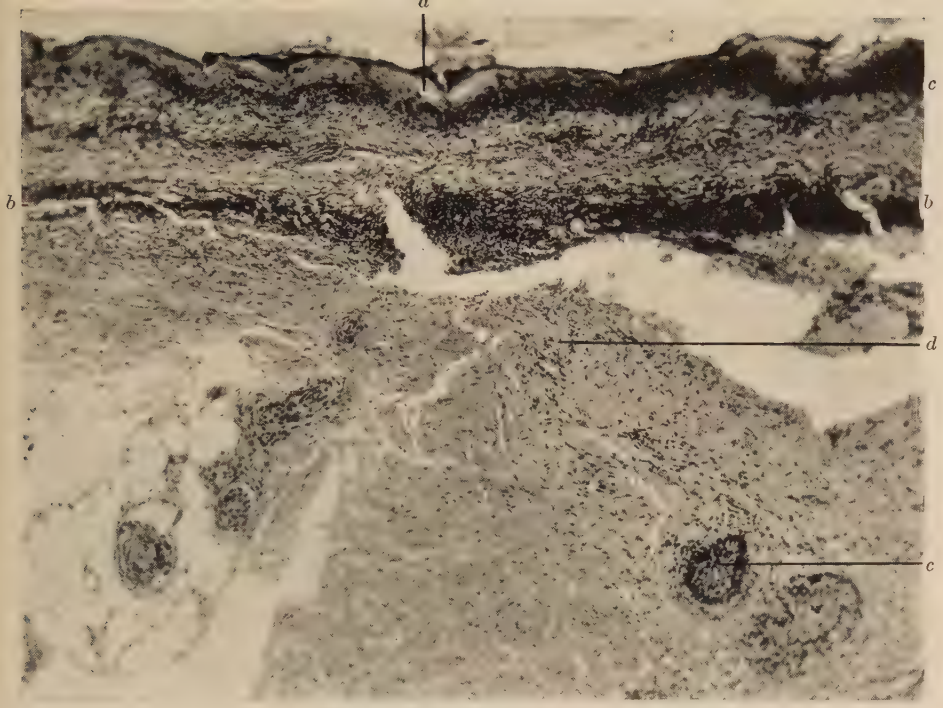

FIG. 11.—Case III. High-powered view. *a*, Tanned membrane. *b*, Separation zone. *c*, Hair follicle. *d*, Connective tissue.

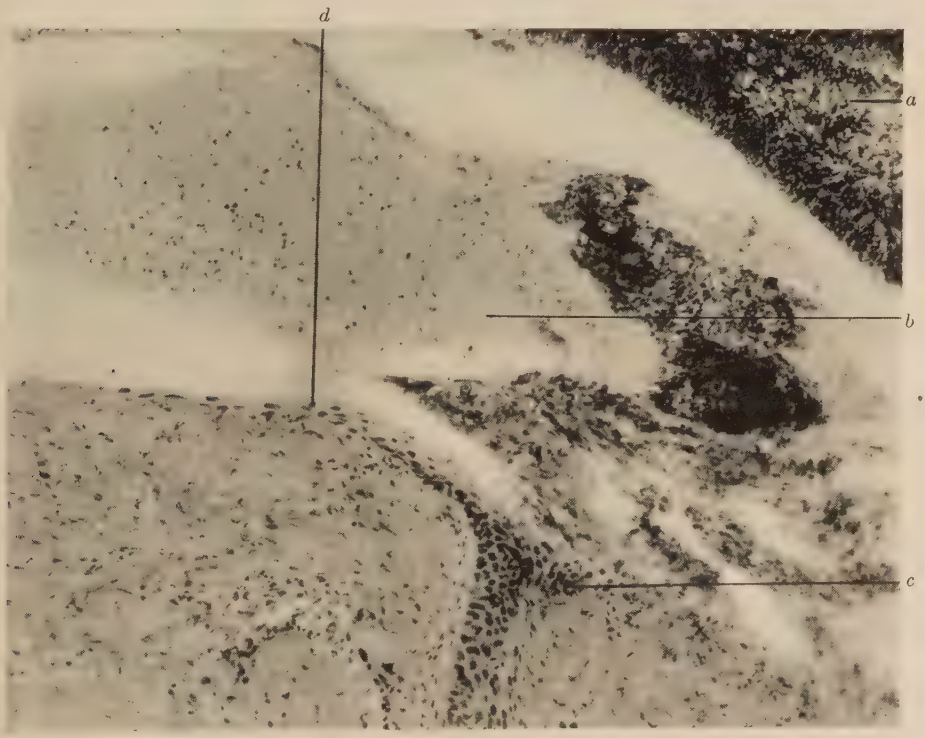

FIG. 12.—Case III. High-powered view. *a*, Tanned membrane. *b*, Separation zone. *c*, Hair follicle. *d*, New epithelium arising from hair follicle spread out over connective tissue. It is assumed that this is beginning epithelialization arising from the hair follicle, while the tannic acid membrane is still *in situ*.

Case I shows almost complete destruction of the epithelium: In places there are islands which are masses of ill-staining nuclei with loss of cell differentiation and occasional epithelial masses resembling pearls; the connective-tissue stroma is edematous and in places necrotic; the fat shows in places death of the nuclei; some of the hair follicles appear relatively normal and others are necrosed. This early débridement undoubtedly removed islands

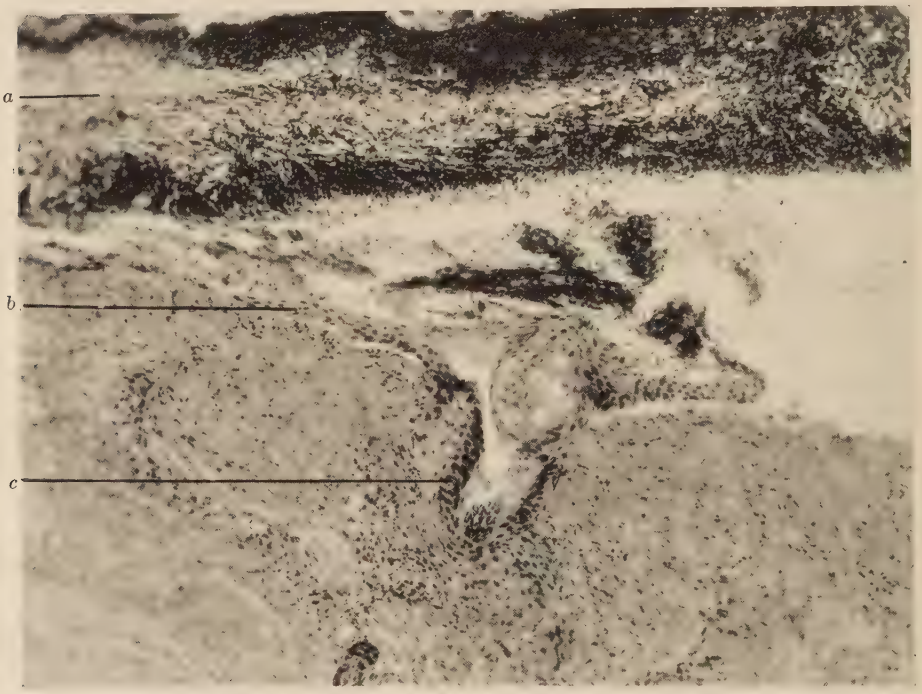

FIG. 13.—Case III. High-powered view. *a*, Tannic acid membrane. *b*, Hair follicle. *c*, New epithelium spread out on connective tissue beneath tanned membrane.

of epithelium in these hair follicles that might have been sources for later regeneration of the skin. Bloodvessels have their walls intensely damaged and there is apparently a coagulative necrosis of the blood. One can realize from this section the tremendous amount of absorption of toxic products.

Case II, after eight days one sees a condition like a sequestrum in osteomyelitis; an area of necrosed tissue separated from the living tissue by a zone of leukocytes. In this specimen the sub-

cutaneous tissues and fat are markedly infiltrated with leukocytes and infection appears to be present. This was taken from the patient who showed such marked improvement following débridement.

In sections made from one case, a child, aged ten years, four days after treatment with tannic acid, the tanned membrane with its beginning separation zone is shown. In the high-power view, a thin layer of flat epithelium extending from the hair follicle is seen spread out over the connective tissue. We assume that this is new epithelium, beginning repair, originating from the hair follicle while the membrane is still in places attached. This proliferated epithelium is a strong argument in favor of the tannic-acid treatment as opposed to débridement. We believe also that this tissue injury is less than that shown in the cases treated by débridement. This, however, might be attributed to the intensity of the burn.

There is still some confusion in the minds of a great many relative to the incidence of ulcers in the duodenum or in other parts of the intestinal tract following cutaneous burns. From our review of the literature we are unable to find any reference to fresh duodenal ulcers found at autopsy.

Bardeen and Weiskoten have made particular studies in quite a number of burn cases dying within a few hours from their injury, and also in late cases, both in adults and children. The intestinal tract contained many punctate hemorrhages and occasional small ulcers in the jejunum and the ileum. Weiskoten emphasized the profound destruction and characteristic appearance of the adrenals. Three of our cases that died had marked distention, suggestive of intestinal lesion; but unfortunately, due to the fact they were coroners' cases, we were unable to perform autopsies.

Tannic Acid. For about six months we have used tannic acid for local treatment, as described by Davidson. The skin is first carefully cleansed. A 2.5 to 5 per cent aqueous solution of tannic acid is then applied as a wet dressing for twenty-four hours. When the burned area becomes a mahogany-brown the dressing is removed and the patient is treated with heated dry air. Usually a tent is erected over the bed and electric lights inserted. The patient is kept as surgically clean as possible. Davidson has propounded the theory that tannic acid coagulates protein and precipitates the poisonous materials in burned tissue, thereby preventing their absorption.

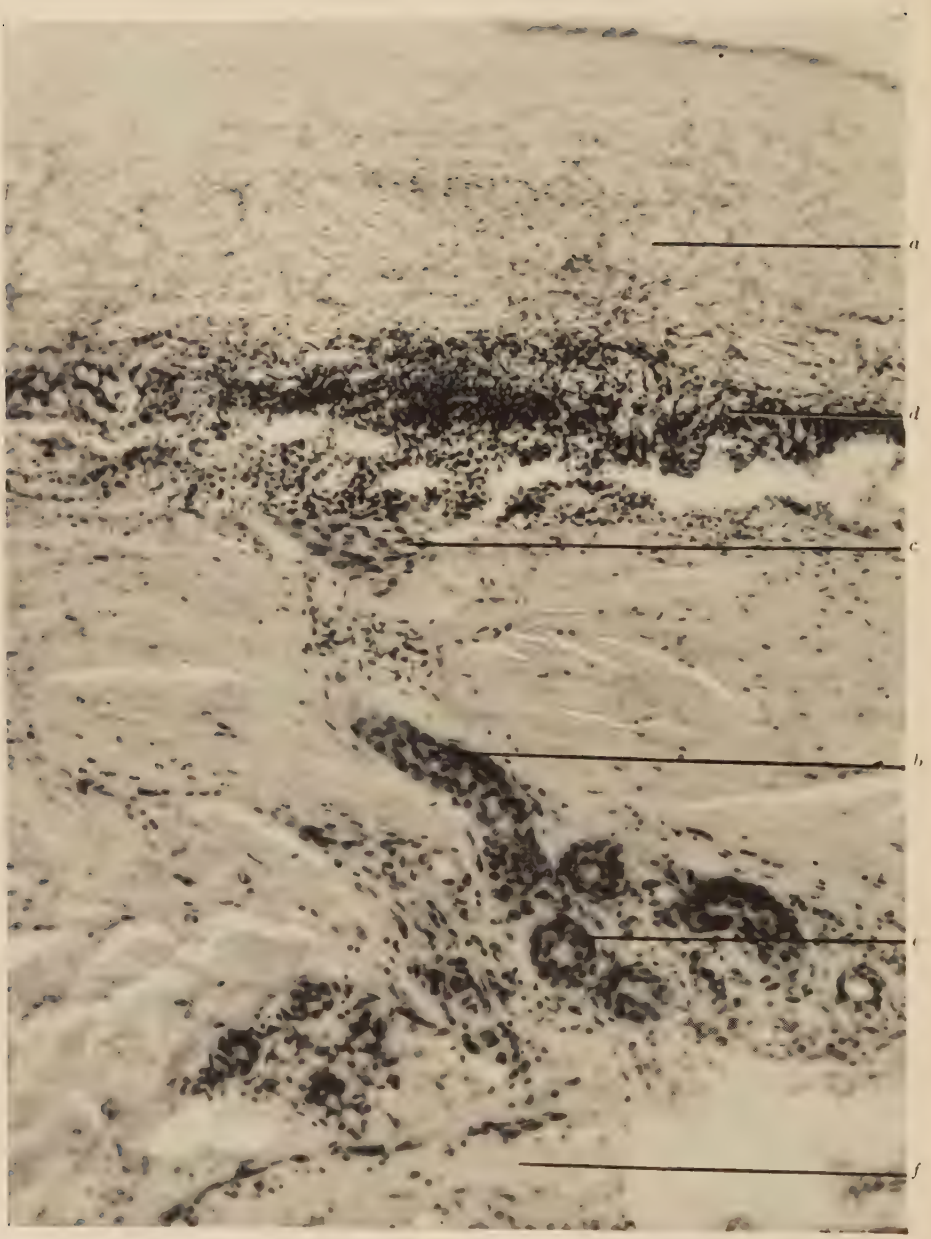

FIG. 14.—Case IV. Section removed from child, aged nine years, treated with tannic acid on admission. Section removed four days after burn, because child was running a temperature and exhibited signs of infection. *a*, Tanned membrane. *b*, Hair follicle. *c*, New epithelium, spread out irregularly on connective tissue. *d*, Exudate of polymorphonuclear leukocytes. *e*, Sweat glands showing infiltration of polymorphonuclear leukocytes and evidence of infection. *f*, Small capillary containing numerous polymorphonuclear leukocytes.

In practice patients treated by tannic acid have a firm mahogany membrane in the burned area; healthy skin is apparently unaffected by the treatment. The tanned area has a leathery consistency. Unquestionably there is less pain associated by this method of treatment than by any other procedure we know. We are all used to seeing a patient suffer intensely the first few days after a burn: Blisters that fill and refill after puncture, dressings that have to be changed daily with agony to both patient and surgeon alike. This is completely changed with tannic acid. The patient is in relative comfort for the first seven to ten days and may continue so until cured if the burn be merely a first or second degree. The membrane should be left to separate of itself, epithelialization occurs beneath it in superficial burns: often attempts at early separation cause bleeding and with bleeding comes infection.

Tannic acid seems to us the best method at present for the treatment of burns—it relieves pain, apparently diminishes absorption of toxic products and makes the care of patients much easier from all points of view. In 2 cases of complete circular burns of the extremities in children the rigid membrane caused by tannic acid produced edema of the distal parts and longitudinal incisions to the fascia were necessary to release the tension.

With first- and second-degree burns epithelialization has often occurred by the time the slough has separated. We have had some deaths at three to four weeks in third-degree burns, where, as will be shown in our analysis of deaths, we believe infection occurred beneath this membrane.

Analysis of Cases. It is impossible to compile statistics of any value in a small series of burns. Too many extraneous factors must be considered, as, for instance, the age of the patient, the patient's general condition before the trauma, the amount of nerve shock due to the injury itself, and, finally and most important, the amount of surface area burned.

In this study we have attempted to accurately estimate the burned area according to enclosed tables (see pages 276 and 277). In reviewing a series of cases that have come in as emergencies, there are undoubtedly many inaccuracies and probably we have overestimated the burned surfaces. We have, however, tried to check this up as carefully as possible. Our results show that the mortality per cent and the length of hospital stay have been improved since the introduction of tannic acid.

Two cases treated with tannic acid: A woman, aged thirty-

two years, with burns of approximately 50 per cent body surface, and a woman, aged forty-two years, with burns of approximately 50 per cent body surface involved, died twenty-four and seventeen days respectively after injury. In both cases the membrane over the first- and second-degree burns had separated, leaving healthy, epithelializing surfaces. The temperatures rose four or five days preceding death and the patients appeared septic. We believe that their lives might have been prolonged had we removed the sloughs covering the third-degree portions, and treated the granulating surfaces by Carrel-Dakin or wet antiseptic dressings.

Treatment of Contractures *vs.* Early Skin Grafting. We have had two very depressingly bad results in circular burns of the thighs and legs in young children, where we attempted to apply extension by Bryant overhead traction to prevent contractures at the knee-joints. Before we realized what was happening, pressure necrosis occurred at the anterior surface of the ankle-joint, followed by deep sloughs. In 1 case marked lymphatic obstruction with edema of the feet resulted. These children are markedly devitalized and do not stand any type of traction. We believe that contractures, where there are circular burns, should be prevented by nurse and doctor frequently during the day applying careful manual traction. Davidson, in Detroit, and Burdick and Beekman, of the Children's Surgical Service at Bellevue Hospital, have advocated early skin grafting as soon as sloughs separate and we are convinced that this procedure should be given precedence over the attempt to correct deformities. If skin grafting is delayed, there is a formation of unhealthy anemic granulation tissue with a scar-tissue base. In the 2 cases where we attempted traction in lieu of skin grafting, we have had to débride this scar tissue before we could have any success with skin grafts. Moreover in 1 case where we grafted early and had contractures at the knee-joint, we obtained a perfect result by anesthetizing the patient and extending the knee. Epithelial and fascial tears in the popliteal space were created during this procedure and immediate skin grafts applied with a successful outcome. We now have an excellent anatomic and functional result.

Blood Chemistry. The urea-nitrogen content and blood-sugars have been taken in a considerable number of these cases. We have found a slight preliminary rise of the blood-sugar within normal limits. Urea-nitrogen has in general been lower than

normal, save where death was imminent. Never has it been higher, however, than 15 mg. per 100 cc. of serum.

CONCLUSIONS. 1. The use of tannic acid is a distinct advance in the treatment of cutaneous burns.

2. The systematic treatment of fluid depletion is of great value.

3. Patients with third-degree burns often have late elevation of temperature and exhibit signs of infection. Life may be prolonged in these cases by removing the tanned dead tissue and treating the infection.

4. Skin grafts should be applied soon after sloughs separate.

5. In children with circular burns of the extremities care should be exercised in attempting to correct deformities.

6. In deep burns of small-surface area immediate débridement associated with skin grafting may be of value.

TABLE I

Total number of cases		104
Deaths:		
Early—within twenty-four to thirty-six hours	13	
Late	17	
	—	30
Percentage mortality		28+%

AGE INCIDENCE OF BURNS

Decades.	1	2	3	4	5	6	7	8
No. cases	36 34%	9 8%	17 16%	20 19%	11 10%	7 6%	1 −1%	3 2%

AGE INCIDENCE OF DEATHS

Decades.	1	2	3	4	5	6	7	8
Early	8	..	1	1	...	..	..	3
Late	6	..	1	3	4	2	1	
Total	14 46⅔%	.. ..	2 6⅔%	4 13⅓%	4 13⅓%	2 6⅔%	1 3⅓%	3 10%

Comment Table I: The high incidence and mortality of children under ten years should be impressed upon the public to stimulate education of mother and child.

TABLE II.—CASES TREATED BY DÉBRIDEMENT

Total number of cases	7
Cases improved or cured	3
Cases that died	4
Average length hospital stay (all cases)	92 days
1 case still in hospital	126 days
Mortality	57 per cent

Deaths: One child, aged two years, 25 per cent of body surface involved, débrided on third day after admission, died that day. One child, aged six days, 35 per cent of body surface involved, débrided three days after admission, died twenty-nine days later. One child, aged four and a half years, 20 per cent body surface involved, débrided two days after admission, died on seventeenth day—bronchopneumonia and dilatation of heart (autopsy record). One adult, aged fifty years, 35 per cent of body surface involved, débrided day after admission, died twenty-four days later (toxin).

TABLE III.—CASES TREATED BY TANNIC ACID

Total number of cases	9
Cases improved or cured	7
Cases that died	2
Average length hospital stay (all cases)	30 days
1 case still in hospital	120 days
Mortality	22 per cent

Deaths: One case, aged thirty-two years, 56 per cent body involved, died twenty-four days after admission. One case, aged forty-two years, 50 per cent body involved, died seventeen days after admission.

Comment Table III: It will be seen that the mortality is less and the hospital stay shortened in cases treated with tannic acid.

TABLE IV.—CASES TREATED BY ELECTRIC CRADLE

Total number of cases	38
Cases improved or cured	19
Cases that died	19
Average length hospital stay (all cases)	21 days
Mortality	50 per cent

Comment Table IV: Cases were treated by this method prior to January 1, 1925. The mortality has been diminished with the later methods.

TABLE V.—CASES TREATED BY OTHER MEANS

Boric acid—Picric acid—$NaHCO_3$—$MgSO_4$ alone or in combination with electric cradle.

Total number of cases	50
Cases improved or cured	45
Cases that died	5
Average hospital stay (all cases)	13 days
Mortality	10 per cent

Comment Table V: As will be seen by Table VII, these were cases of small surface area and very little therapy was required to prevent mortality.

TABLE VI.—AVERAGE HOSPITAL STAY OF RECOVERY CASES TREATED BY ALL METHODS

Débridement	104
Tannic acid	50.2
Electric cradle	39.9
Miscellaneous	14.2

TABLE VII.—AVERAGE PERCENTAGE AREA INVOLVED IN RECOVERY CASES

Débridement	28.3 per cent
Tannic acid	30.0 per cent
Electric cradle	19.7 per cent
Miscellaneous	11.2

DISCUSSION

DR. EDWARD P. RICHARDSON, Boston, Mass.:

Since the appearance of Dr. Davidson's article, last summer, the use of tannic acid followed by exposure under the electric cradle has been the routine treatment for burns in the Massachusetts General Hospital. Our impressions are that the results in regard to mortality are better than with any form of treatment previously used in the hospital. There is one very definite advantage, it seems to me: The trea ment forms a hard membrane, the surfaces remain dry, serum loss does not occur and the dehydration which contributes to the immediate mortality is less likely to occur in these cases.

URINARY OBSTRUCTIONS IN CHILDHOOD*

By CHARLES G. MIXTER, M.D.
BOSTON, MASS.

Urinary obstruction is unconsciously associated with the mental picture of the later years of life. One visualizes the old prostatic with the distressing train of symptoms following back pressure and diminished renal function. This clinical picture is well understood and adequately handled surgically. Obstruction of the urinary outflow in infancy and early childhood on the other hand, has rarely been recognized except at postmortem examination. The frequency of its occurrence and its menace to health and life in early years has not been appreciated.

Urinary stasis is sooner or later followed by infection. Unless the obstruction is relieved and adequate drainage established, it is practically impossible to eradicate the infection and ultimately one or both kidneys are destroyed by pyelonephritis. The importance of the underlying factor of urinary obstruction in childhood is little realized and many of the cases are seen only when the destructive process is far advanced. A complete urological examination can and should be made at any age when signs of urinary retention are present or when there is persistent or recurrent pyuria.

Briefly the routine of examination has been as follows: After the usual urinary, renal function, and blood chemistry studies, a cystogram is made with 20 cc. of 12 per cent sodium iodide in both the anteroposterior and lateral positions to ascertain the presence of a ureteral reflux diverticulum or dilatation of the posterior urethra. A cystoscope is then passed, both ureters are catheterized and pyelograms are made. The catheters are then withdrawn to the lower ureter and ureterograms are made. The ureters are then drained and a final film is taken. This will

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show whether there is delayed emptying of the pelvis or any portion of the ureter. The divided renal-function test has been unsatisfactory as the catheters used are necessarily so small that there is urinary leakage around them, vitiating the test. Cystoscopy can usually be carried out in girls over seven or eight years of age and boys over eleven years, under local anesthesia. In younger children a general anesthetic is required.

Usually children are referred for urological examination for one of four conditions; pyuria of long standing, acute attacks of abdominal pain of unexplained origin that may be referred to the kidney region, difficulty in urination, and hematuria. The symptoms and physical signs of obstructive lesions are so indefinite that an accurate diagnosis is practically impossible without the aid of the cystoscope and uretero-pyelograms. Where vesical retention is present, spina bifida occulta or other lesion interfering with bladder innervation, must be ruled out.

Leaving out of consideration phimosis and stricture of the external meatus, there are three regions of the urinary tract especially prone to obstructive lesions in early life, namely, the uretero-pelvic junction, the vesical insertion of the ureter and the region of the verumontanum in the male urethra. More commonly such lesions are congenital, rarely they may be acquired.

Stricture of the uretero-pelvic junction is followed by hydronephrosis. Inflammatory changes are usually found on microscopic examination of the sections taken through the point of stricture. Where sufficient renal tissue is present in spite of prolonged back pressure, a plastic on the uretero-pelvic junction is the operation of choice.

Congenital stenosis may occur, which in rare instances may be bilateral. Large cystic tumors are formed which can be identified by the thin shell of kidney substance spread out on the surface of the cyst. So little functional tissue is present, that nephrectomy is almost invariably indicated.

Faulty insertion of the ureter into the renal pelvis at too high a level, will cause inadequate drainage. The greater the distention of the pelvis, the sharper the angulation at the uretero-pelvic junction and the more complete the obstruction. Where the hydronephrosis is slight and there is little evidence of renal destruction, a plastic on the uretero-pelvic junction or section and reimplantation of the ureter into the most dependent portion of the

pelvis, should be performed. Frequently, however, the kidney is so far destroyed by back pressure and infection that the indication for nephrectomy is evident.

The ureter in early childhood is more tortuous than in the adult and ureteral kinks near the pelvic junction are common. It is difficult in any given case to determine just how far the angulation revealed by ureterograms may be responsible for urinary stasis and secondary infection. That a kink can be the cause of urinary obstruction was clearly demonstrated in a recent case. Nephrectomy was done for a large hydronephrosis, the size of a grapefruit. The sharply kinked ureter was sectioned several inches below the pelvis. On removing the clamp the hydronephrotic sac remained distended but by traction on the sectioned end of the ureter the angulations were eradicated, there was a free flow of urine and the distended pelvis rapidly collapsed.

At the vesical end of the ureter, stricture of the orifice with or without cystic dilatation may occur. Stricture with cystic dilatation in one instance was bilateral. In another patient, a boy, aged sixteen months, there was unilateral reduplication of the ureter and pelvis. One of the orifices was normal, the other emptied on the surface of a cystic dilatation. The cyst was of large size and involved the base of the bladder to such an extent that it occluded the internal urethral opening, producing vesical retention.

The intravesical partition of such cysts is thin, consisting of an external and internal lining of epithelium and an intermediate layer of connective tissue. Excision of this partition completely relieves the obstruction and is more adequate than simply laying open the cyst, which proceedure may be followed by cicatricial stenosis. Cystoscopic operative methods are impossible through the small calibered instrument usually required for cystoscopy in childhood. Where fulguration can be employed, destruction of the vesical wall of the cyst can be accomplished satisfactorily by this means, if the dilatation is of small size.

Where stricture of the orifice is present without cystic dilatation, slitting the intramural portion of the ureter from the opening upward for a distance of about $\frac{1}{2}$ inch through a transvesical exposure, and drainage of the dilated and usually infected ureter with a good-sized soft rubber catheter for ten days to two weeks, has secured a very satisfactory result.

Two views are held regarding the etiology of these strictures: (1) That they are of congenital origin; (2) that they occur as the result of infection. In view of the early age at which they may be encountered and the absence at times of evidence of marked infection on urinary examination, the former hypothesis would seem the more probable.

Diverticula may produce urinary retention from obstruction at the vesical neck. Situated usually in the vicinity of a ureteral orifice, they may dissect downward behind the base of the bladder as they enlarge until the distended sac shuts off the internal meatus. They are of rare occurrence in childhood, comprising approximately 5 per cent of the total number of bladder diverticula reported, and those causing retention constitute only a fraction of this small number. In our clinic for the past three years a routine cystogram has been taken in all cases of bladder retention or where the symptoms are those of difficulty in urination. No instance of diverticulum however has been encountered and none is found in the past records. Nevertheless it should be borne in mind as a possible cause of retention in childhood.

Obstruction in the male urethra occurs from congenital valve-like folds in the region of the verumontanum or from hypertrophy of the verumontanum itself. More rarely there may be a true congenital stricture. Recently 56 instances of valve formation have been collected from the literature and in 1923, 7 examples of obstruction from hypertrophy of the verumontanum were reported from the autopsy protocols of the Babies' Hospital in New York. These figures undoubtedly give a very inadequate idea of the frequency of occurrence of congenital urethral obstructions. The routine method of performing postmortem examinations does not include removal of the deep urethra with the bladder and upper urinary tract. It is only in very recent years that attention has been called to these lesions, since which time the number of cases observed in our clinic would suggest a higher incidence than these figures apparently indicate.

Two instances of congenital stricture of the deep urethra have come under observation. This anomalous condition arises from failure of the downward growing proximal portion of the urethra to unite accurately with the inward growing distal segment during development. In the cases observed each segment ended blindly, save for a minute opening connecting them laterally, where the

blind ends overlapped. The condition was clearly demonstrated by open perineal operation in one instance; in the other a coudé filiform passed the stricture and was followed by gradual dilatation with relief of bladder distention and marked general improvement.

Causes of Urinary Obstruction in Childhood

			No. of cases.
A. Intrinsic lesions of urinary tract	1. At uretero-pelvic junction	*a.* Stricture uretero-pelvic junction . . .	7
		b. Faulty insertion ureter into pelvis (with hydronephrosis) . .	2
		c. Kinks of ureter (with hydronephrosis) . .	6
	2. At vesical insertion	*a.* Stricture of ureteral orifice	3
		b. Cystic dilatation of orifice	2
	3. In posterior urethra	*a.* Congenital stricture	2
		b. Valves at verumontanum	6
		c. Hypertrophy of verumontanum . . .	0
B. Condition producing obstruction by pressure from without	1. Accessory renal vessels		6
	2. Reduplication of pelvis and ureter .		7
C. Stasis produced by primary dilatation of ureter; no demonstrable obstruction	1. Megaloureter		5

The pathology produced in the urinary tract above the obstruction is striking. The bladder wall is greatly thickened with hypertrophy of the musculature causing trabeculation, such as is seen in the cystoscopic examination of the prostatic bladder. The ureters are tortuous and dilated and there is bilateral hydronephrosis. The uretero-vesical sphincters are frequently incompetent, permitting a ureteral reflux to the kidneys, and a simple cystogram will often give a satisfactory uretero-pyelogram.

Removal of the obstruction may be accomplished by fulguration of the valve or hypertrophied verumontanum. In infants this

procedure is not possible and usually in childhood suprapubic cystotomy or perineal section with excision is necessary.

In addition to the intrinsic obstructive lesions of the urinary tract in childhood, there are encountered two congenital anomalies producing urinary stasis by pressure on the ureter from without. These are accessory vessels running to the lower pole of the kidney and reduplication of the renal pelvis and ureter.

Accessory renal vessels may be either artery, vein, or both and commonly the artery arises from the aorta and the vein empties into the vena cava. The vessel crosses the ureter at right angles near the uretero-pelvic junction and even slight mobility of the kidney may angulate and obstruct this point. This type of anomaly is not uncommonly bilateral. Should a case having typical renal colic from intermittent hydronephrosis disclose at operation an accessory lower polar vessel as the cause, the opposite kidney should be carefully studied. Even if there had been as yet no symptoms referable to this kidney, should the pyelograms demonstrate a delayed emptying time either in the prone or upright position, or should beginning hydronephrosis be evidenced by slight bulging of the inferior concave outline of the pelvis, exploration of this kidney should be seriously considered.

In the very early stage of intermittent hydronephrosis before the normal elasticity of the renal pelvis is lost, the pyelogram will show no deviation from the normal outline. A girl, aged eight years, was admitted to the hospital with the history of three attacks of right-sided abdominal pain during the preceding six months. The story and clinical findings were not characteristic of appendicitis. The urine was negative. Ureteral catheterization revealed nothing abnormal through there was incomplete filling of the right pelvis. There was no distention of the pelvis and the calices were not clubbed. Operation did not seem indicated and while the child was awaiting her discharge from the hospital, there was an acute attack with severe pain, chill, fever, vomiting and a tense palpable tumor in the right kidney region. This mass disappeared within four hours. On exposure the kidney and pelvis appeared normal but there was an accessory lower polar vessel. This was tied and sectioned and in the two years since operation there has been no recurrence of the attacks.

Section of the accessory renal vessel is not unattended by danger. A considerable segment of the lower pole of the kidney

may be solely dependent on this vessel for its circulation. An example occurred in a girl having recurrent attacks of right renal colic. Following division of an accessory lower polar vessel for intermittent hydronephrosis, there was a slight persistent serous

FIG. 1.—Faulty insertion at ureteropelvic junction. No. 28860. Female, aged ten years, July 15, 1920. Ureteropyelograms show advanced bilateral hydronephrosis. Greatly delayed emptying time of pelvis. Radiographic appearances suggest a faulty insertion of the ureter into the renal pelvis. Not confirmed by operation.

discharge from the wound and some irregular elevation of temperature. After five weeks, a necrotic cast of about one-fourth of the kidney, entirely detached from the remaining renal tissue, was removed from the wound. The sinus rapidly healed.

Before tying off the vessel, the circulation should be temporarily

obstructed for several minutes and the lower pole observed. Should marked cyanosis occur, simple nephropexy or nephropexy

FIG. 2.—Kink of ureter. No. 88898. Female, aged eight years, November 17, 1925. Ureteropyelogram shows marked hydro-ureteronephrosis on the right side, sharp kink at the uretero-pelvic junction. Cause of acute attacks of hydronephrosis. Exploration advised and refused.

combined with suture of the ureter in a position to avoid angulation may be attempted. In advanced dilatation, division of the

ureter and anastomosis with the pelvis at its lowest point, below the level of the aberrant vessel, should be attempted in preference to jeopardizing the viability of any considerable portion of the renal tissue.

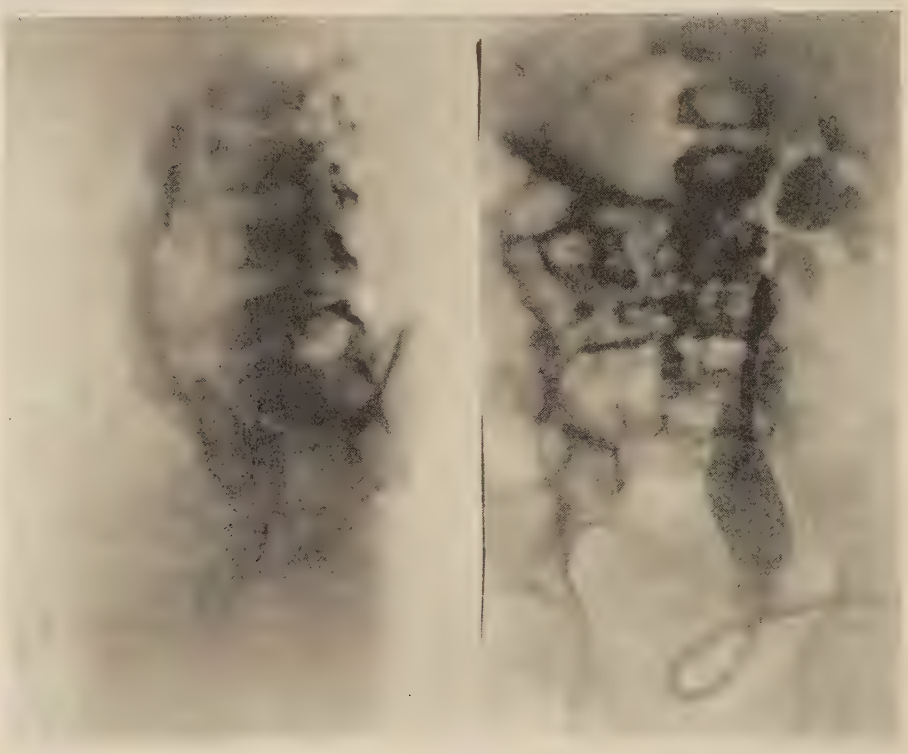

FIG. 3.—Stricture of ureteral orifice No. 54441. Female, aged seven years, February 12, 1925. Postoperative radiogram. Before operation attempts to catheterize right ureter failed. Orifice would not admit the finest catheter. Cystotomy and plastic on right ureteral orifice with drainage. Tight stricture found at orifice causing attacks of hydronephrosis and dilatation of ureter.

Reduplication of the pelvis and ureter may be unilateral or bilateral and may be complete or incomplete. Where the duplication is complete the ureter from the upper pelvis empties by way of the lower of the two orifices in the bladder, while the ureter from the lower pelvis runs to the upper orifice. This necessitates a twining of the ureters about one another and where they cross

obstruction is apt to occur. Where duplication is incomplete, fusion of the two ureters may take place at any point between the

FIG. 4.—Cystic dilatation of ureteral orifice. No. 88255. Female, aged eight years, October 29, 1925. Child uremic and died without surgical intervention. Autopsy specimen revealed a large cystic tumor at the site of the right ureteral orifice and a similar small cyst on the left. With a syringe and considerable pressure, no fluid could be forced through the stenosed orifices on the surface of the cysts after removal of the specimen.

lower pelvis and the bladder. Obstruction from the pressure of one ureter on the other is less likely to occur than in the complete variety, but either type is frequently accompanied by circulatory anomalies that may produce stasis.

Frequently duplication of the pelvis and ureter causes no stasis and gives rise to no symptoms. Attacks of pain from intermittent hydronephrosis or persistent pyuria from chronic pyelonephritis are the indications that call for urological investigation. The operative treatment demanded varies with the individual case. Freeing the ureter and ureteropexy may be sufficient to ensure

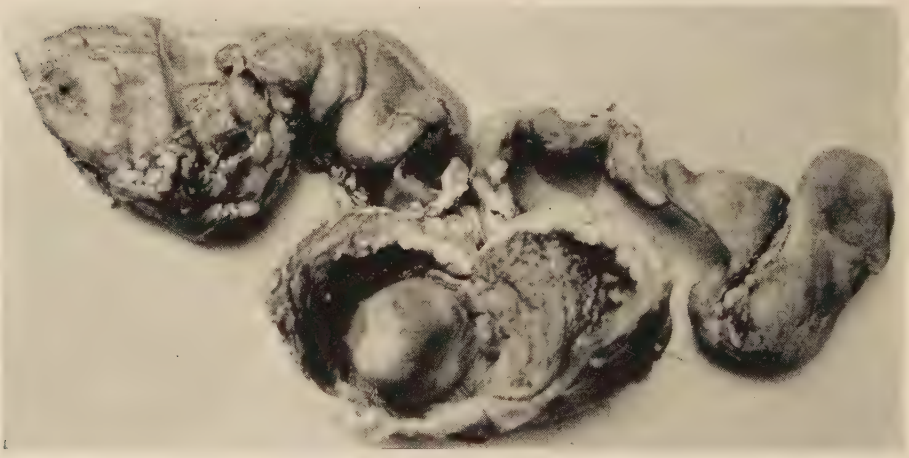

FIG. 5.—Autopsy specimen obtained from case referred to in Fig. 4, showing bilateral stricture of ureteric orifices with cystic dilatations. Bilateral hydro-ureteronephrosis.

proper drainage. Division of some unimportant elements of the anomalous circulation, or section of one ureter and an end-to-side anastomosis of the proximal end into the other ureter may eliminate the stasis. Heminephrectomy might be attempted where the renal destruction is sharply limited to one-half the kidney. Finally, nephrectomy may be demanded by advanced hydronephrosis or pyonephrosis.

Perhaps the most interesting condition met in childhood producing urinary stasis and destructive renal back-pressure, is the enormous dilatation of the ureter called megaloureter, idiopathic dilatation and giant ureter. It is frequently bilateral. Although

it might not properly be classed as an obstructive condition, as no stenosis of the ureteral orifice is present, it is evident that the aperture of the normal orifice is of insufficient caliber to adequately drain the tremendously distended ureter and pelvis present in these cases.

The etiology is still in doubt. It is most generally held to be a congenital anomaly, in many respects similar to megalocolon. However in the latter condition the intestinal wall shows a great

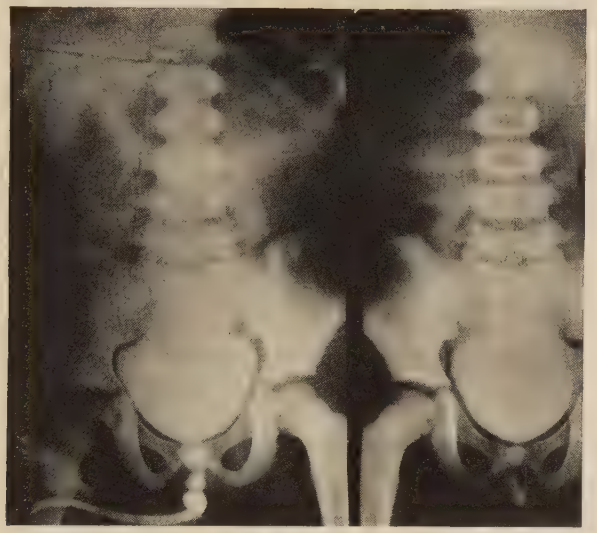

FIG. 6.—Congenital stricture of urethra. No. 45254. Male, six years, January 14, 1925. Cystogram shows a dilated posterior urethra. The distal urethra is outlined. The intermediate area is bounded distally by the strictured portion of the anomalous urethra and proximally by valves at the level of the verumontanum. Confirmed by perineal section.

increase in the muscular layer. In the few specimens of megalo-ureter that we have had the opportunity to study microscopically, the thickening of the ureteral wall has been due to fibrosis and inflammatory infiltration with very little increase in the muscular elements. Defective innervation of the ureter diminishing the muscle tone and permitting dilatation, has been advanced as an explanatory theory. In the absence of any associated impairment of innervation elsewhere it would seem improbable. No lesion of the lower urinary tract causing obstruction can be

demonstrated and for this reason it is maintained that a dilatation secondary to a more distal obstruction cannot occur. Infection, for example a long-standing pyelonephritis, has been advanced as the primary factor, the ureteral dilatation being a

FIG. 7.—Urethral obstruction from congenital valves. No. 68121. Male, aged seven years, February 28, 1924. Ureteropyelograms show enormous bilateral hydro-ureteronephrosis. Bladder trabeculated. A valve type of obstruction was demonstrated in the posterior urethra by suprapubic cystostomy.

secondary manifestation. This view would not seem probable as many cases are seen where the urinary examinations over long periods of time show evidence of severe infection and yet the pyeloureterograms are essentially normal. On the other hand in

occasional instances of megaloureter, the infection may appear to be relatively slight. A mechanical explanation is conceivable due to the obliquity of the course of the ureter through the bladder

FIG. 8.—Urethral obstruction from congenital valve formation. I. H. No. 10593. Male, aged six weeks, April 26, 1923. Autopsy specimen shows obstructing valves running to verumontanum. Effects of back pressure are seen in the thickened trabeculated bladder, the tortuous, dilated ureters and the bilateral hydronephrosis with renal destruction.

wall causing angulation or valve action at the uretero-vesical junction. Ureteral stasis could induce hydroureter and a vicious cycle would be established, so that the greater the dilatation the more complete would be the obstruction.

Several cases of megaloureter in boys have been observed where at first sight no obstructive lesion was apparent. More careful study however revealed an obstruction in the posterior urethra.

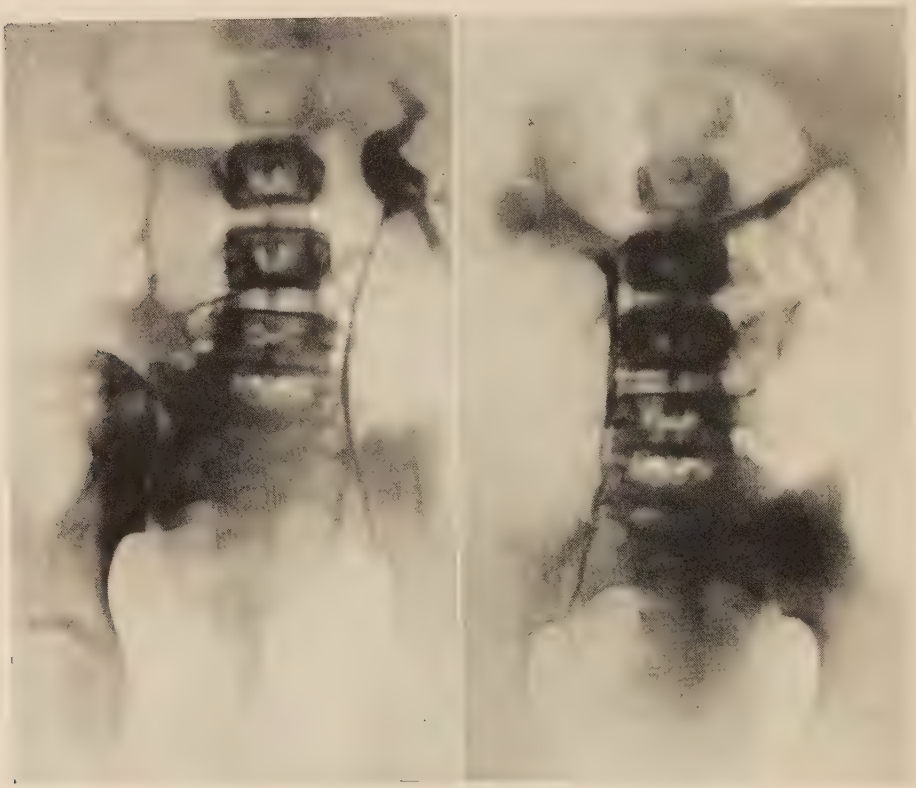

FIG. 9.—Accessory lower polar vessel. No. 36211. Female, aged ten years, April 26, 1926. Ureteropyelograms show marked left hydronephrosis and a normal ureter. There is a suggestion of slight hydronephrosis on the right side and the emptying time of the pelvis was delayed. Function of left kidney so greatly diminished it could not be estimated. Left nephrectomy revealed an accessory vein to the lower pole causing obstruction and consequent hydronephrosis and infection. Possibility of a similar anomaly on the right side.

The findings in the bladder and upper urinary tract in these cases did not differ from those in which no such mechanical obstruction could be demonstrated. Even in the short female urethra valve-like folds of mucous membrane might occur that could obstruct

and yet be very difficult of demonstration. In those cases where vesical retention does not occur, faulty implantation of the uretero-vesical junction could readily produce the ureteral dilata-

FIG. 10.—Operative specimen removed from case shown in Fig. 10. Hydronephrosis produced by accessory lower polar vessel.

tion. Mechanical obstruction with a secondary infection would, therefore, appear adequate to explain cases of megaloureter.

With megaloureter the effects of long-continued absorption and toxemia are evident. The children are underdeveloped and under-

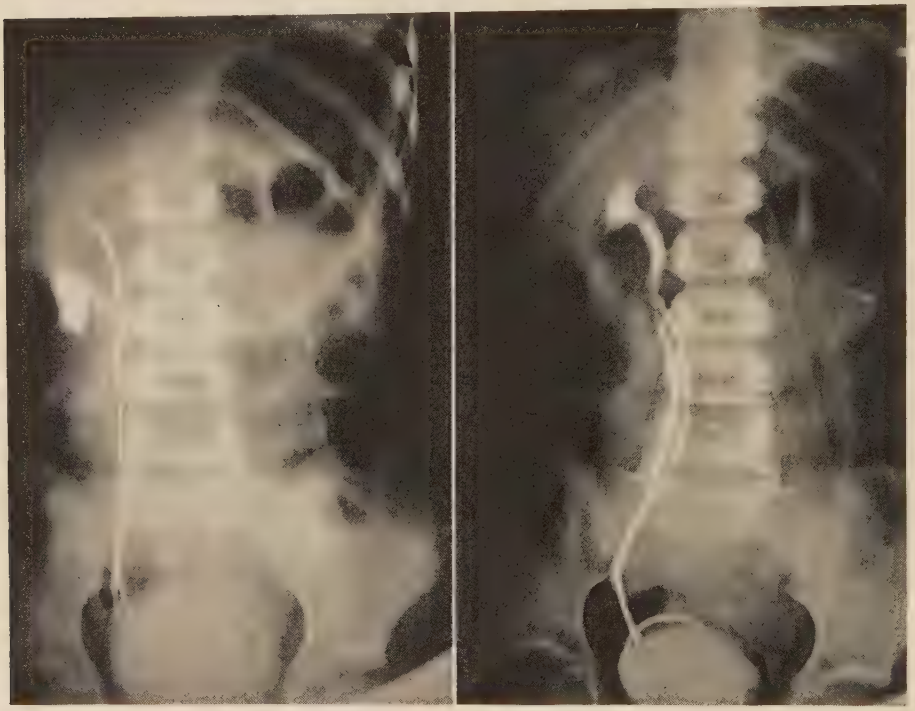

FIG. 11.—Reduplication of pelvis and ureter right. No. 77171. Female, aged six years, November 19, 1924. Ureteropyelograms show slight dilatation of lower pelvis of the right kidney. Little blunting of calyces. Both ureters dilated from ureteropelvic junction to point where they cross. Delay in emptying time. Obstruction caused by crossing of ureters, producing acute hydronephrosis. Attacks relieved by operation at which an anomalous renal circulation was demonstrated.

nourished and may show mental retardation. Renal efficiency is markedly impaired and uremic symptoms may develop. In one case multiple bilateral calculi were present. These were thought to be the result of infection and urinary stasis, and not the cause of the megaloureters.

Cystoscopy reveals a cystitis with injection of the mucous membrane and trabeculation similar to that seen in cases of excessive hydro-uretero-nephrosis where a definite posterior urethral obstruction can be demonstrated. In the cases investigated, the ureteral orifices have appeared normal and no inkling has been given of the condition present in the ureter. In the literature however a number of instances are reported in which the ureter orifices were of the "golf-hole" type, greatly dilated, the

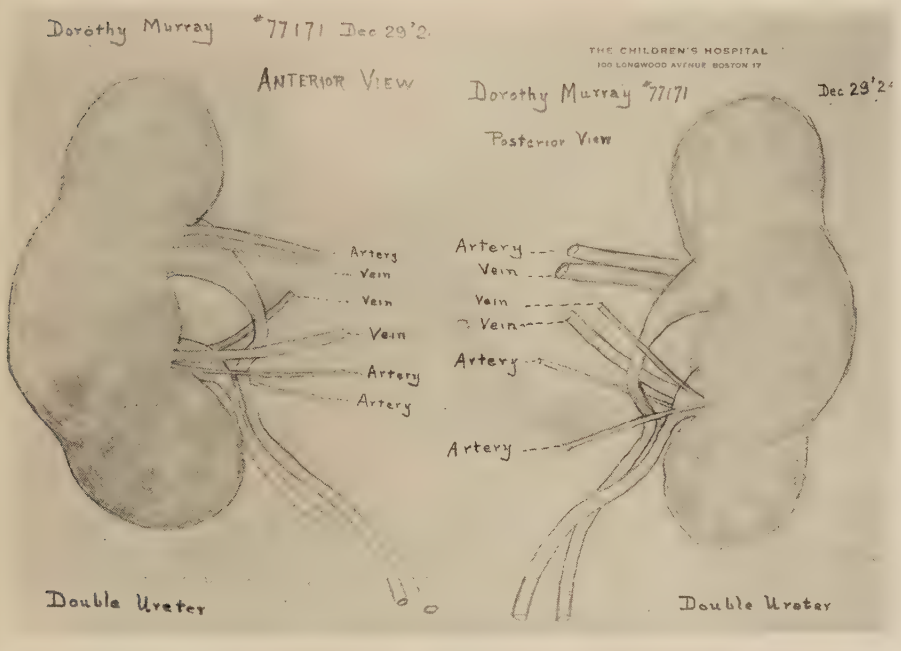

FIG. 12.—Schematic drawing of anomalous circulation found at operation in case of double ureter.

margins stiffened, and suggestive of the openings of diverticula. On insertion of the ureteral catheter a free regular flow of many cubic centimeters of urine is obtained. Ureterograms show enormously dilated and at times tortuous ureters, the dilatation extending from the bladder to the uretero-pelvic junction. At this point the caliber is usually somewhat narrowed. There is distension of the renal pelvis and at times this does not seem

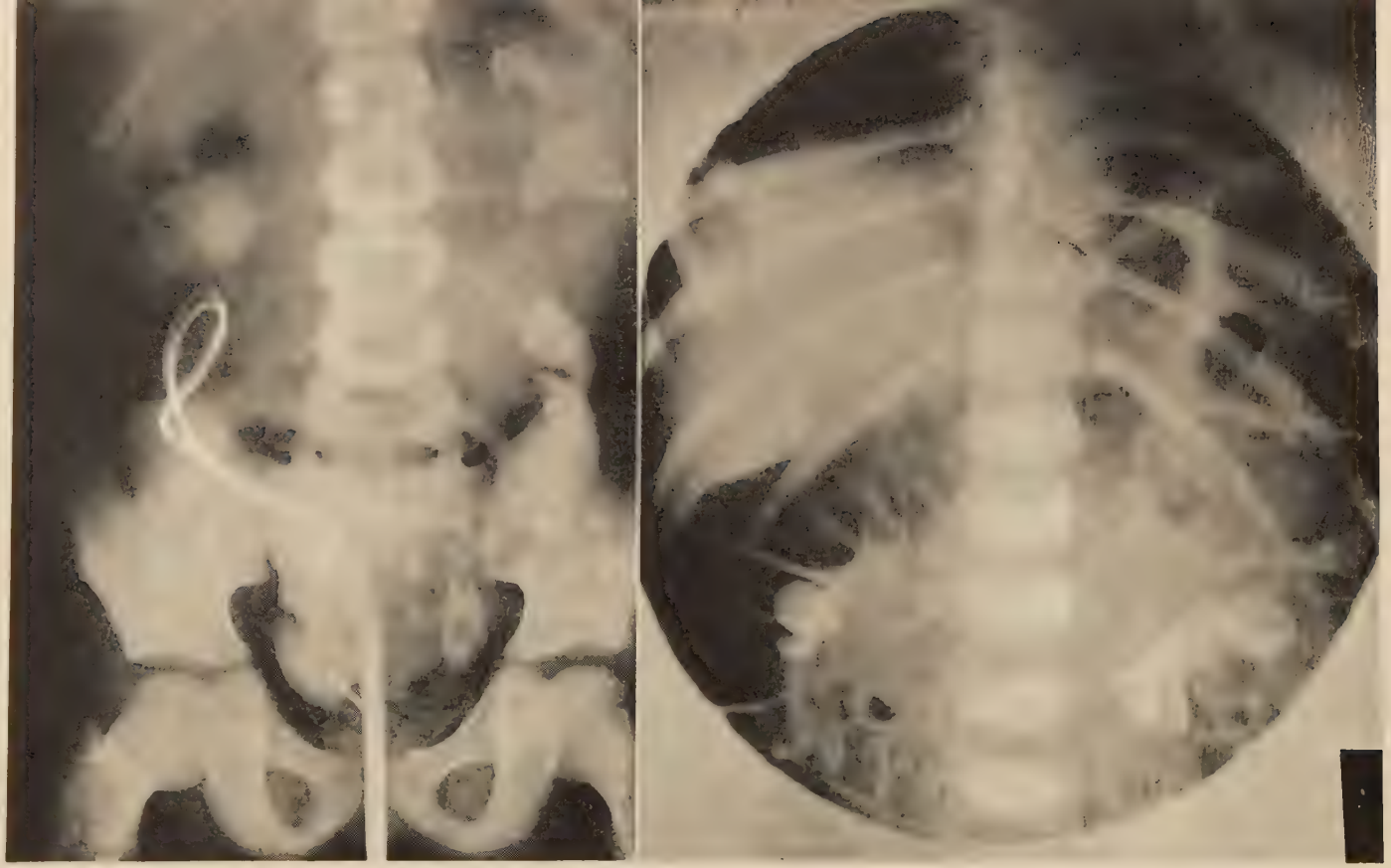

FIG. 13. Megaloureter. Bilateral nephrolithiasis. No. 24222. Male, aged seven years, January 15, 1924. Ureteropyelogram shows enormous bilateral dilatation of the ureters and hydronephrosis. Calculi were present in the lower ends of both ureters and the pelves of both kidneys. Two years after the last operation for drainage of pyonephrosis and removal of multiple calculi, the patient died of uremia. No merchanical obstruction was demonstrated at autopsy, but unfortunately the urethra was sectioned below the bladder without

commensurate with the degree of hydroureter present. The calices are mushroomed and there is advanced renal destruction.

The operative treatment of megaloureter has been unsatisfactory owing to the extensive renal damage previously sustained. A plastic on the ureteral orifice seems indicated, as the size of the normal opening is inadequate to drain the tremendous hydroureter above, which may attain the size of the large bowel. Unfortunately a cystostomy with slitting of the orifices, accompanied by tube drainage of the kidneys through the ureters, is sooner or later followed in many instances by lighting-up of the infection and anuric symptoms. Consequently bilateral nephrostomy must be considered in many of these cases before undertaking any operative measures on the ureters. Renal efficiency has been so far reduced by back-pressure and infection that the margin of safety is very narrow. Infection with progressive renal impairment is practically impossible to check in the absence of a good urinary current through the dilated ureter and it may well be that a permanent double lumbar nephrostomy will be found to give the most comfort and the longest expectation of life. However, the importance of a thorough and painstaking search for an obstructive lesion should be emphasized, as it seems probable from our experience that such a lesion may exist in a considerable number of the cases that have previously been classified as idiopathic dilatation of the ureter.

SUMMARY. 1. Obstruction of the urinary outflow in childhood is far more common than has been generally recognized in the past.

2. Urinary stasis is followed sooner or later by infection and progressive renal destruction from back pressure and inflammatory changes.

3. Urinary obstructions are chiefly due to congenital anomalies and may be caused by intrinsic lesions of the urinary tract or by pressure from without.

4. Intrinsic lesions are situated most commonly at the ureteropelvic junction, the vesical orifice or the region of the verumontanum in the male urethra.

5. Causes of pressure from without on the urinary tract are usually due to anomalous renal vessels or reduplication of the pelvis and ureter.

6. Megaloureter produces urinary stasis and renal impairment.

The etiology is in doubt but it seems probable that an obscure obstructive lesion, either in the posterior urethra or at the uretero-vesical junction, associated with a secondary infection, offers sufficient explanation. It is generally bilateral. The ureteral orifices are normal but are not competent to drain adequately the enormously dilated ureters.

7. In any type of lesion encountered it is imperative to relieve the obstruction to the urinary outflow at an early stage to prevent extensive renal destruction.

8. The destructive process is already far advanced in the majority of the cases before an attempt is made to establish an accurate diagnosis.

9. Cystoscopy, cystograms and uretro-pyelograms are indicated in childhood in all cases suggesting obstructive urinary lesions or showing persistent or recurrent pyuria.

THE "REHABILITATION" OF THE SURGICAL PATIENT THROUGH BIOCHEMICAL METHODS, WITH SPECIAL REFERENCE TO DIABETES*

By WILLIAM J. MAYO, M.D.
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In 1850, Jakob Moleschatte, Professor of Physiology, Anatomy and Anthropology in the University of Heidelberg, said, "Man is composed of the food he eats." Poor Moleschatte: his statement was regarded as blasphemous, and he was expelled from his positions, and from the university. In the seventy-five years that have elapsed, the truth of his deduction has been recognized, and we can congratulate ourselves that intolerance and religious bigotry have abated sufficiently to permit discussion of the science of nutrition as applied to man.

Fundamentally, "rehabilitation" concerns preservation of the body; biochemically it is directly related to the metabolism of foods. The newer studies on nutrition have given the key to many problems concerning general physical conditions which heretofore have been unsurmountable. Studies of the blood and of the blood plasma as well as of the tissues of the body have usually made it possible to restore resistance and overcome toxemia preoperatively. There has been much confusion in the nomenclature with regard to toxic conditions; the types of toxemia often being named as though they were caused by the vital organ which shows the final lethal change, for example bronchopneumonia or terminal nephritis, rather than the fundamental causes which actually bring about dissolution.

The rehabilitation of surgical patients through biochemical methods before operation is perhaps the most important consideration in modern surgery. The object is not only to improve the patient's general condition, but to overcome the results of disturbed function, the deficiency of vital substances, and the formation or

* Read by title.

retention of poisonous excretory products. Unless the function of vital organs can be restored nearly to normal in serious diseases that do not immediately threaten life, the risk of surgical operation may be so great as to outweigh the advantages to be obtained. Walters states facts as follows:

"Life depends on the oxidation of carbon. Carbohydrates are used as glucose, stored as glycogen in the liver, and the excess deposited in the body as fat. Unfortunately, fats cannot, to any considerable extent be reconverted into glucose; furthermore, sufficient glucose must be available to maintain combustion of fats; otherwise the higher fatty acids will not undergo complete combustion, and acetone and diacetic acid will appear in the tissues and cause toxemia, which in the more severe grades may terminate in coma, as in cases of diabetes.

"On the contrary, proteins can, to a considerable extent, be converted into glucose, but they contain nitrogen. Under stress, as in cases of starvation or hepatic disease, sufficient glucose is not available; the protein tissues of the body are broken down for the purpose of producing the necessary glucose, and the resultant excess nitrogen must be excreted in the urine. The kidneys may be unable to excrete this excess of nitrogen, which then accumulates in the blood, for the most part as urea, and a small percentage as creatinin, producing toxemia of which uremia is a manifestation."

Diabetes presents two problems to the surgeon: (1) The cure and prevention of diseases requiring surgical treatment, which arise directly or indirectly from diabetes, and (2) safety in the performance of necessary operations on patients with diabetes for lesions not associated with the diabetes.

Long ago Pavy pointed out that the mere reduction of the amount of sugar in the urine was not of itself a sufficient safeguard for patients obliged to undergo surgical procedures, and that restriction of carbohydrates in the diet should not be carried to a point at which the patient's general health and strength suffered. Billings and Allen have still further illuminated the treatment of diabetes by demonstrating that if the carbohydrates in the diet are reduced too greatly, the nutrition being maintained by proteins and fats, diabetic coma and other manifestations of severe diabetes are likely to ensue, but that if the patient is treated by the so-called starvation method, the fats and proteins being reduced as well as carbohydrates, this danger will be greatly lessened.

In 1915, Berkman demonstrated that the surgical patient with diabetes who was too thoroughly prepared for operation by over-reduction of carbohydrates, did not withstand operation as well as the underprepared patient. He advised maintaining the patient's nutrition by a mixed diet. This was practised in the clinic with the result that the former high mortality rate following operations on patients with diabetes was greatly reduced.

Before insulin became available, Wilder and Adams, by careful preoperative and postoperative dietetic treatment of patients with diabetes, were able to reduce the mortality rate following operations in selected cases practically to that in cases without diabetes. In such cases at least 100 gm. of carbohydrates is now given for five days before operation and not less than 75 gm. on the average each day after operation, with sufficient insulin to maintain tolerance. Recently Wilder and Adams reported 141 unselected cases in which major operations were performed for general surgical diseases, complicated by diabetes, with but four deaths, a record which would be excellent in cases uncomplicated by diabetes. With the aid of insulin, operations of expedience may be performed on patients with severe diabetes on whom in the past only operations of necessity would have been undertaken.

At the finish of a recent Marathon race the condition of the runners was noted with special attention to the state of the blood. The runners who finished the race, for instance Nurmi, showed a normal content of blood-sugar, whereas the runners who dropped exhausted by the way, often in a shock-like condition sometimes resembling mild grades of coma, showed a low content of blood sugar. By eating sugar and resting in the horizontal position they quickly regained strength. Later some of these runners were given sweet chocolate to eat as they ran, and these maintained a higher degree of endurance. The efficacy of sugar in maintaining endurance has been demonstrated also by the Arabs, who eat dates on long marches.

One of the greatest commanding officers of the Civil War was Stonewall Jackson, who, with a small army, was able to march his troops so rapidly in the region across the Potomac from Washington that they were called "foot cavalry." He accomplished this by marching the soldiers several hours, then having them lie flat and relax for a few minutes. By resting in this manner the lactic acid, which accumulates in the muscles during action and produces fatigue, is in large part restored to glucose, as A. V. Hill

and Meyerhof have recently shown. The soldiers were then given whatever food the sutler wagons afforded, usually carbohydrates, with fluids; this quickly supplied the necessary sugar, and they were again marched a shorter distance with another period of rest and similar restoration. By repeating this process at intervals, Jackson was able to move his ragged, barefoot men over bad roads as much as forty miles in twenty-four hours and have them in fighting condition. One might say that Jackson prolonged the Civil War two years; he was able to keep Washington in fear of capture, forcing McDowell's army to remain on guard, instead of aiding the offensive on Richmond. It was believed that if Washington were taken by the Confederates, England and other foreign countries might recognize the belligerents of the South.

The test for the amount of sugar in the urine as a guide to the severity of diabetes has largely given way to a test for sugar in the blood. Sugar in the blood is a threshold substance. The threshold varies greatly in height in different persons. When the sugar exceeds a certain level, so to speak, it flows over the top and appears in the urine. A person with a low-sugar threshold may have glycosuria after eating an unusual amount of carbohydrates, such as candy, and yet not become afflicted with diabetes. Another person with high-sugar threshold may have no sugar in the urine, but an excess in the blood, which in cases of slight infection may cause serious disturbances. Even study of the sugar in the blood may fail to tell the whole story. Sugar is stored in the liver and other tissues as glycogen, which is merely a condensation product of glucose.

Experience in the clinic would indicate that certain patients with a normal amount of sugar in the blood may in times of stress be unable to store sufficient glycogen in the liver, as is illustrated by the following case:

The patient, a woman, aged forty-four, had had malignant disease of both ovaries and tubes, for which abdominal hysterectomy was performed. She left the operating table in good condition, but six hours later coma developed, no clue to which had been given in the history or preliminary physical examination.

Examination of the blood showed a very low sugar content and acidosis. An intravenous injection of 10 per cent, glucose solution with bicarbonate of soda and a moderate amount of insulin was given. The patient regained consciousness in a few moments. The injections were continued for three days, and convalescence

was rapid. The patient left the hospital in due time, and has remained well.

Wilder reports two cases of carcinoma of the liver in which hypoglycemia occurred postoperatively with accompanying shock-like symptoms which were relieved by intravenous injections of glucose. Patients with hyperthyroidism are susceptible to hypoglycemia, particularly if insulin is used, probably an exhaustion of the blood-sugar from overwork caused by the high metabolic rate.

The study of the comatose conditions which accompany diabetes is most interesting. The cause of the coma lies in the metabolism of the fats. The metabolism of fats and their combustion, producing heat, energy for vegetative functions, and notably water, take place very slowly. This explains the state of certain animals during hibernation, when their temperature drops to about half that normal to warm-blooded animals, and there is a general abeyance of function, while life at a low level is sustained by the slow oxidation of the stored-up fat. Unfortunately, without the addition of glucose, fat cannot be completely burned. When there is a lack of available sugar, fat which can be converted into sugar if at all, does not exceed 10 per cent.

The molecule in animal or vegetable fat always has an even number of atoms, which are oxidized in the body two at a time. When the carbon atoms are burned down, say to four, if sugar is not available, diacetic acid or acetone is formed, and coma may result. If the molecule of fat contained an uneven number of carbon atoms which burned two at a time, the low point would be five, at which valeric acid would result, or three, at which lactic acid would result, both harmless. An attempt to produce a fat with an uneven molecular composition has been successful in a chemical sense, but unsuccessful from the physical standpoint, in that the body seems to be unable to metabolize such artificial fats. When glucose is not obtainable from ingested carbohydrates or proteins, and the stored glycogen in the liver is exhausted, glucose can only be obtained by breaking down deposit or tissue proteins. About 58 per cent of these proteins can be converted into glucose, but the process liberates into the blood stream an excess of nitrogen, recognized as urea, and disorders result, not only from acetone, but from the rising tide of blood-urea. Diabetic coma, formerly so greatly feared by physician and surgeon alike, yields readily, almost specifically, to insulin. A properly regulated

diet, with judicious employment of insulin and glucose solution renders the patient with diabetes a good surgical risk in the majority of cases. But care is always necessary, since with improper handling the mildest forms of diabetes still may become a grave source of danger.

It is interesting, as related to the pancreatic theory of diabetes, that when children have died in diabetic coma, ordinary methods of necropsy often disclose no changes either in the pancreas or in the islands of Langerhans. On the other hand, Allen states that in such cases necropsy made immediately after death, while the body is warm, does disclose changes in the islands of Langerhans. Pancreatic changes are usually manifest in older persons who, generally speaking, are overnourished, not always obese, but nearly always large eaters. Perhaps the demand on the pancreas beyond the nutritional necessity causes the body insulin to wear out and results in functional deficiency of the islands of Langerhans.

If pancreatitis accompanies infection of the gall-bladder, it does not take the form of atrophy of the pancreas and of the islands of Langerhans, rather characteristic of diabetes. On the contrary, the changes in the pancreas, as a result of biliary infections are usually of the type called chronic pancreatitis, enlargement and thickening. Removal of the infected gall-bladder in the presence of chronic pancreatitis may be followed by improvement in the diabetes, but such a favorable result is not obtained with sufficient frequency to justify operating, in the hope of curing the diabetes, on a gall-bladder suspected of being diseased. At least it is a comfort to know that diabetes is not necessarily a contraindication to an operation for definite disease of the gall-bladder.

Someone has facetiously called diabetes a foot- and mouth-disease. Food in the form of carbohydrates in excess enters the mouth; in the susceptible person the insulin machinery is exhausted with the result so frequently seen, that the patient dies from gangrene of the foot. It has been a common experience in cases of diabetes, that when there is arteriosclerotic gangrene of the lower extremities, which necessitates amputation of part of the limb, great improvement in the general condition of the patient with a reduction in the symptoms of diabetes may take place after the amputation. One of the necessary steps in the treatment of diabetes is to eradicate all sources of focal infection which can be removed safely. Joslin is wise indeed in his advice to amputate the leg above the knee in cases of the acute, florid type of diabetic

gangrene when there is discoloration of the entire foot and ankle without a line of demarcation, and high temperature. This is a condition which in the past has so frequently resulted in death, and in which early amputation will save life. Joslin emphasizes the fact that care of the feet, next to a proper dietetic regimen, is the most important measure in the treatment of diabetes. A patient with diabetes should wear several pairs of shoes in succession. The trimming of corns and calluses should be regarded as a serious operation, to be done under antiseptic precautions, in the morning rather than at night, so that all swelling and edema in the muscles about the foot from the day's usage will have disappeared.

Carbuncles and boils on the back of the neck are most common in men, especially those with diabetes, and usually in those who have the back of the neck shaved. It is quite probable that the present fashion, no doubt a good one, of women cutting their hair, particularly shaving the back of the neck, may reduce this masculine predominance. Clipping the hair on the neck should be safer for the patient with diabetes than shaving it.

The use of the actual cautery to destroy carbuncles, advised by Charles H. Mayo in 1901, is extraordinarily efficacious. It has been used in the clinic successfully for many years for all forms of carbuncles, both in the patient with diabetes and the patient without diabetes. The dark-red heat of the soldering iron is best, and with it the entire area of the carbuncle is converted into an eschar. The eschar is left without a dressing, and the dry treatment for burns is applied. If necessary, an electric fan is used to keep the surface dry, and dry powdered boric acid may be rubbed in to absorb moisture. When the slough begins to separate, the usual applications of antiseptic wet dressings are made. Astonishing results are seen in cases of severe carbuncle. The patient suffering with pain and high temperature, within a few hours after the cautery treatment is quite comfortable and up and about with normal temperature.

In closing it may be said that progress in the surgical treatment of patients with diabetes has come, not so much through improvement in technic, as in the preoperative care of the patient, in the attention to such small matters as the care of the feet, the prevention of skin infections, and in the intelligent use of diet, insulin and glucose.

SOME CLINICO-PATHOLOGIC ANALOGIES BETWEEN THE BILIARY TRACT AND THE URINARY TRACT*

By EDWIN BEER, M.D.
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A STUDY of the similarities in the processes that take place in rather unrelated systems is liable to be of more than passing interest and may prove instructive and stimulating. Even though our knowledge be but surface gleanings, and though future investigations may upset much that we believe in, still certain striking analogies between pathological and clinical observations in the biliary and urinary tracts have struck me and have interested me for years, and to some of these I wish to refer in this very brief paper. I feel that a better appreciation of these analogies has given me a better understanding of the two systems, even though at times speculation rather than complete scientific proof supports the thesis.

There are certain rather self-evident similarities which must be mentioned to be dismissed. At the *fons et origo* of each system is a paired glandular organ—for the liver is much like the kidneys. It is in fundamental structure a fused or bilobed organ—each half, from an excretory standpoint at least, separate from the other. Both liver and kidneys have definite excretory functions and in both the excretion is poured into two ducts—to be stored in the two bladders and subsequently again passed through a duct into the outer world or into the intestine which communicates directly with the exterior.

Passing these anatomical and physiological similarities rapidly and turning our attention to the clinical and pathological, and surely more speculative problems, it must be admitted that despite the vast amount of investigation devoted to the biliary system,

* Read by title.

our knowledge of the urinary tract is far more complete. An intimate knowledge of the latter may throw some suggestive lights on processes that take place in the biliary tract that are less clear and are difficult to understand; and *vice versa*, an appreciation of what we have learned in the study of diseases of the liver and of the biliary tract may assist us in interpreting our observations on the urinary tract.

In studying these similarities between the two systems the subject naturally falls into several subdivisions, and the analogies stand forth rather vividly—perhaps more vividly than they should. For purposes of this discussion I have divided the available material under the following three heads:

1. Cholesteremia and uricemia;
2. Lithiasis: (*a*) Primary; (*b*) secondary.
3. Infections;

and will limit myself to a brief discussion of these, as they illustrate rather cogently the analogies between the two tracts.

1. Cholesteremia and Uricemia. Cholesterol may be considered as a more or less specific hepatic excretion and uric acid bears a somewhat similar relation to renal function, even though both substances are widely distributed in the other body tissues. Both substances are regularly present in definite amounts in their respective excretions and the amount (or percentage) seems to vary with the concentration of these substances in the blood-stream. For many years it was thought that the concentration in the blood-stream could not be altered by diet, and, consequently diet was thought of questionable influence on the amounts of cholesterol or of uric acid excreted in the bile or in the urine. Gradually, experimental feeding experiments have shown that the blood concentration can be increased and with that the amounts excreted are also increased. On the one hand, the cholesterol content can be increased by eating a fatty diet, *e. g.*, egg-yolk, brains, cream, etc.; on the other, the uric acid content can be increased by eating foods rich in nucleins, *e. g.*, thymus, spleen, liver, lungs, kidneys, etc. Moreover, in certain diseases the blood content of these two substances is affected; for instance, the post-infective period of typhoid usually shows an increase in blood cholesterin, and the uric acid content is increased in pneumonia at the crisis. More interesting, however, is the observation of the influence of functional disturbances of the liver and of the kidneys

on the blood concentration of these two substances. In bilateral renal obstruction such as we see in early prostatic cases, the early evidence of functional impairment of the kidneys is found in an increase of uric acid in the blood. Similarly, in the biliary tract, one of the first signs of choledochus obstruction, incomplete or complete, is a heaping up of the cholesterol in the blood. In both cases, with removal of the cause the physiological disturbance is relieved and the blood chemistry returns to normal.

Whether in such and similar functional disturbances there are irregular fluctuations and that from minute to minute (so to speak) the concentration in the blood varies, is not known, though it is probable. The occasional spilling over of concentrated solutions, of oversaturated colloidal solutions, might account for many of the obscure clinical pictures that have masqueraded here and abroad under various appellations, *e. g.*, dysfunction of the gall-bladder, kinks of the cystic duct, strictures of the ureter and the like. In both the urinary and the biliary tract, not infrequently typical colics take place simulating attacks of lithiasis, and most careful study and most searching operative investigation fail to find a stone or any evidence of acute inflammation. In some cases these attacks occur after removal of the gall-bladder and cannot be ascribed to adhesions or to the abdominal wall giving away. Aschoff says we must now recognize that the occurrence of dyskinetic attacks of cramp in the gall-bladder region are more frequent than was formerly thought.

The study of some of these cases in the urinary tract has proven of great interest and it is possible that an analogous explanation may apply to the somewhat similar picture in the biliary tract. The urine is a complex solution containing many ingredients in solution. Recent work has suggested that many of these are in a supersaturated solution by virtue of the action of the colloids. Pauli and Samec similarly showed that calcium phosphate and calcium carbonate in albuminous solutions are seven times more soluble than in water. If, then, a change in the colloidal system takes place, the salts (*i. e.*, crystals) will naturally be thrown out in the urinary passages, and what is called a colic, due to a "shower" of uric-acid crystals passing down the ureter, will occur. On voiding such urine the crystals will be in solution again provided the colloid is reversible, and, if allowed to stand, in the course of a day or two the nebecula (colloid) separates and the uric acid

crystals are again apparent as crystals. The process inside the body is probably an instantaneous one—totally dissimilar from that seen in the test-tube. I have seen this instantaneous change in urines as they have passed through the female urethra—the patient passing a white opaque urine, due to sudden dropping out of phosphates, while the residual urine immediately obtained by catheter was yellow and clear. Similarly, in the upper tract, I have apparently induced a colloidal change by passing a ureter catheter and induced a unilateral fresh phosphaturia, and from the second side obtained clear urine such as was found originally in the bladder.

These phenomena have as yet been studied too little, and to apply the same methods to the biliary tract has been impossible. The presence of numbers of cholesterin crystals in the duodenal secretion or in the gall-bladder in colic cases in which no stone or inflammation was found, might establish the existence of a process analogous to that seen in the urinary tract. But even without this evidence the therapeutic test offered by a change in diet suggests that similar colloidal disturbances are present in both systems, and that the overconcentrated solutions of uric acid in the one and of cholesterol in the other are at fault. Restriction in protein intake controls the attacks of uric-acid showers, and restriction in fats seems to have a similar effect in many of these biliary cases.

2. Lithiasis. Turning to the question of lithiasis, or stone production, in these tracts, despite our imperfect knowledge, striking analogies are readily discerned. For many years the teachings of Naunyn held the field and gall-stones were considered the result of the combined activities of bacterial infection and stagnation of bile. In a study of these factors, published some twenty years ago, it appeared to me that possibly a third factor—which might be a disturbed hepatic metabolism—was an essential element. Since then Aschoff has rather successfully separated the pure radial cholesterin stones from the whole group and emphasized the fact that they owe their origin to a faulty metabolism of the liver. He again called attention to the fact that these stones are usually found in bladders in which there is no sign of infection or of past or present inflammation. Apparently the cholesterin is sedimented in the slowly moving bile of the gall-bladder and the crystalline stone forms. In the urinary bladder of prostatics,

previous to the period of infection, a very similar sedimentation is favored in the residual urine and in these cases we often encounter uratic stones. In both cases, possibly a temporary spilling over of the stone material, an increased excretion, may contribute to produce the colloidal instability that leads to the precipitation of the stones. In the kidney such primary stones are more difficult to study; but here in the kidney pelvis and calices are encountered uric acid, oxalate, cystin, xanthin and indigo stones which have appeared to develop without the interference of infective agents, possibly due to processes analogous to those underlying the formation of the pure cholesterin stones. The analogy between the uric acid (and uratic) stones and the cholesterin stones is very striking. Owing to the obscurity of the metabolic processes underlying the other types of renal stones just mentioned, one must hesitate in grouping them together too intimately.

When, however, infection sets in, a totally different type of stone develops and these secondary stones are quite typical. In the biliary tract we find the cholesterin pigment calcium stones, and in the urinary tract (both in the kidney and in the bladder) the various more or less hard phosphatic (ammonium magnesium phosphate and calcium magnesium phosphate) stones. These may contain a nucleus of the primary type and are usually built up in layers and have a network of proteid material derived from the inflammatory exudate in which they developed. Thus it is apparent that there is a striking analogy in the formation of the primary as well as of the secondary postinfection stones in both the biliary and the urinary tracts.

3. Infections. In studying the behavior of these two extensive excretory systems, it is quite striking to see how readily the bacteria (toxins?) are passed through these glands without any apparent damage to their parenchyma. For many years it has been known that in bacteriemas the offending organisms are excreted in the urine. Apparently a very similar process takes place in the liver and biliary system, for under such circumstances in the bile a great variety of bacteria have been recovered. This transit through these two systems seems to do no permanent damage unless foreign bodies or obstructive conditions obtain and then local inflammatory changes may be induced.

In the kidneys bacillary and coccic infections are very common

and many of these produce such mild local symptoms that they are liable to be overlooked. Many such infections have been misinterpreted. Undoubtedly in the majority of the cases an almost complete repair and restoration of function occurs. Do analogous infections occur in the biliary system? Some writers believe infections of the liver are "extraordinarily" common (Naunyn, Poppert, Aschoff). If they are common, owing to the lack of local symptoms and laboratory evidence, we are surely not recognizing them in the clinic, and further attention must be directed toward their diagnosis. Undoubtedly, the liver has marvellous recuperative power, and, being a remarkably silent organ, many a febrile disease may be misinterpreted when the pathology lies within this organ.

The repeated attacks of infection in the kidney parenchyma eventually lead to the peculiar scarred kidney of old pyelonephritics, and if similar processes occur in the liver, one cannot but wonder whether some of the changes of the cirrhotic liver are not developed in this same way.

In closing this very inadequate study of some of the more striking similarities between the biliary and urinary tracts, one cannot but feel apologetic for attempting to put in words much that is still in the developmental stage. The parallelism has impressed me as too striking to be passed by, and I am sure it will stimulate further work and lead to a clearer understanding.

A TWELVE-YEAR STUDY OF THE FOLLOW-UP IN MALIGNANT DISEASE*

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Five hundred and seventy-three cases of malignant disease were observed in the First (Cornell) Surgical Service during the period February, 1913, to January, 1925.[1]

An attempt was made to follow every case for at least ten years, but rapid changes of residence, departure from the city or country, etc., often nullified our work. We were materially aided by the efficient and devoted coöperation of the Social Service Department in charge of Miss Josephi.

No sadder report of the disheartening status of cancer surgery has come to our attention. It is, however, inevitable, dealing only with facts (Table I).

Table I.—All Operations Whether Radical, Palliative or Exploratory.

Operative Cases	
Number of operations	437
Number of deaths	308
Number of known living	73
Unknown	56
No Operation	
Number of cases	136
Number of deaths	92
Number of known living	12
Unknown	32

* Read by title.

[1] The routine follow-up on these cases was closed in October, 1925; we are constantly receiving additional data through our follow-up service and any information has been added up to date.

TABLE I.—GROUPED AS TO SEX AND AGE

Male, 200. Of this number, 53 were stomach; 33, intestines; 10, epithelioma of lip.

Female, 237. Of this number, 78 were breast; 38, uterus; 27, intestines; 18, stomach.

(Females predominate, although there are fewer beds for females available in the New York Hospital. This fact explains why carcinoma of stomach in females is one-third that in males.)

AGE

Ages.	Number.	Carcinoma.	Sarcoma.	Endothelioma.	Hyperne-phroma and teratoma.
Below 10 . . .	4	1[1]	3	0	0
10–20 . . .	7	3	3	1	0
20–30 . . .	37	16	14	6	0
30–40 . . .	75	56	18	0	1
40–50 . . .	127	111	11	4	2
50–60 . . .	116	110	3	3	0
60–70 . . .	56	54	1	1	0
70–80 . . .	12	12	0	0	0
80–90 . . .	3	2	1	0	0
	437	365	54	15	3

	Per cent.
Carcinoma cases:	
Over 40 years	79
40–60 "	60
Sarcoma cases:	
Under 40 years	70
20–50 "	80

A simplified classification makes large groups of the various pathologic conditions, *e. g.*, carcinoma and epithelioma a single group; all forms of sarcoma another one.

Table II is the keynote of this paper. Of the 437 operative cases that came under our observation only 64 are living today without recurrence and only 13 have survived the accepted but valueless time limit of five years.

To those unfamiliar with the cancer question these results seem to reflect severely on the practice of surgery and the ability of the operators. The great bulk of the work was done by three members of this Association, Gibson, Lee and Hitzrot, whose work may be taken as a fair cross-section of the surgical practice of this country.

[1] Case of neurocytoma of kidney.

TABLE II.—LIVING CASES. NO RECURRENCE. DIVIDED ACCORDING TO NUMBER OF YEARS POSTOPERATIVE

Ten Years.
- Carcinoma, 2.
  - Tongue.
  - Breast.
- Sarcoma, 2.
  - Bone.
  - Gum.

Eight Years.
- Carcinoma, 1.
  - Breast.
- Sarcoma, 2.
  - Intestines.
  - Bone.

Seven Years.
- Carcinoma, 2.
  - Intestines.
  - Uterus.

Six Years.
- Carcinoma, 2.
  - Breast.
  - Face.
- Sarcoma, 1.
  - Bone.
- Endothelioma, 1.
  - Lymph nodes.

Five Years.
- Carcinoma, 7.
  - Appendix, 2.
  - Breast.
  - Face.
  - Lip, 2.
  - Scrotum.
- Sarcoma, 5.
  - Bone.
  - Lymph nodes.
  - Neck.
  - Stomach.
  - Uterus.

Four Years.
- Carcinoma, 7.
  - Appendix, 2.
  - Breast, 2.
  - Stomach.
  - Thyroid.
  - Uterus.
- Sarcoma, 1.
  - Bone.
- Endothelioma, 1.
  - Parotid.

Three Years.
- Carcinoma, 6.
  - Breast.
  - Intestines.
  - Rectum.
  - Stomach.
  - Tubes and ovaries.
  - Uterus.
- Sarcoma, 2.
  - Bone.
  - Forearm.
- Endothelioma, 2.
  - Ovary.
  - Parotid.

Two Years.
- Carcinoma, 7.
  - Breast, 2.
  - Face.
  - Kidney.
  - Submaxillary gland.
  - Uterus, 2.
- Sarcoma, 4.
  - Bone.
  - Tendon sheath.
  - Finger, 2.
- Endothelioma, 1.
  - Lymph nodes.

One Year or Less.
- Carcinoma, 6.
  - Breast, 4.
  - Penis, 1.
  - Face, 1.
- Endothelioma, 2.
  - Lymph nodes.
  - Neck.

Total, 64 cases.

Before adopting too harsh conclusions it should be remembered that this paper is practically an unique contribution to the subject. While the total number is not impressive, I can find no similar study of the *total material* of twelve consecutive years, and on which a vast amount of personal effort, time and money have been spent in the pursuit of results. Similar research emanating from other institutions would doubtless furnish surprises.

Table III shows only a few still alive with recurrence. There were six cancers of the breast recurring after five years.

TABLE III.—LIVING. WITH RECURRENCE

Nine Years.
- Carcinoma.
  - Breast: After 9 years carcinoma in opposite breast; original site of operation all right.

Four Years.
- Carcinoma.
  - Breast, 2.

Three Years.
- Carcinoma.
  - Breast.
  - Stomach.

Two Years.
- Carcinoma.
  - Uterus.
- Sarcoma.
  - Intestines.

One Year or Less.
- Carcinoma.
  - Intestines.
  - Stomach.

Total number of cases, 9.

TABLE IV.—SARCOMATA CASES MORE BENIGN THAN THE CARCINOMA

Of the 64 living cases with no recurrence:	Per cent.
Sarcoma cases living 5 years or more postoperative	58
Carcinoma cases living 5 years or more postoperative	36
Endothelioma cases living 5 years or more postoperative	16

Table V gives the relative freedom from recurrence of the several types. The frightful results in cancer of the intestines and stomach are impressive.

TABLE V.—STATISTICS BASED UPON KNOWN RESULTS ONLY

	Per cent.
Carcinoma of face, living, no recurrence	80
Endothelioma of lymph nodes	75
Carcinoma of appendix	66
Sarcoma of bones	53
Carcinoma of lip	40
Carcinoma of breast	18
Carcinoma of uterus	15
Carcinoma of tongue	14
Carcinoma of intestines	4
Carcinoma of stomach	3

The 100 per cent mortality of the conditions described in Table VI is readily understood, although the 100 per cent mortality in cancer of the gall-bladder may not be generally appreciated. Our 15 cases irrespective of condition or treatment were dead within six months.

TABLE VI

100 per cent mortality: Gall-bladder, esophagus, larynx, liver, pancreas, prostate, peritoneum and omentum

Table VII is based on the known deaths only. The mortality would doubtless be higher if the "unknown" were included.

TABLE VII.—KNOWN MORTALITY

Type.	Number of operations.	Deaths.	Per cent.
Hypernephroma and teratoma	4	4	100
Carcinoma	323	274	84
Sarcoma	47	29	61
Endothelioma	8	1	12

A summary of malignant tumors is appended.

This study is limited to ward cases. In private patients we cannot pursue the same rigid tracking down of patients, taking every possible means of obtaining information. It seems unwise, therefore, to include in the same group cases conforming to different standards. Our private patients, particularly the breasts, show rather better results.

This investigation aims to give a broad survey of the mass results of malignant disease. Therefore, we have omitted the dissection of the statistical evidence into the finer distinctions, as recommended by some investigators, because of the confusing element of personal judgment of conditions. We do not think it wise to rate the unknown as necessarily dead, as quite a number of these fall in the class giving better results.

No branch of surgery shows such statistical fallacies as the results of cancer. Probably the great bulk of the cases operated radically have existing metastases which lie dormant. For example Case 223299, living seven years after resection of the colon, had also diffuse metastases of the lymph nodes as proven by microscopic examination. A private patient with suture of a free perforation of a gastric cancer with extensive metastases

(microscopic examination) is alive two and a half years after operation. Case 240695 came in with acute obstruction from a sarcoma of the jejunum with extensive infiltration of the mesocolon. A resection of the gut and an end-to-end anastomosis was done, although the section went through the invaded area. The patient lived three years and seven months after the operation.

Another private patient with bilateral carcinoma of the ovaries is now living seven years after a second operation, nine years after the first operation, although at the last operation there were diffuse metastases throughout the abdomen. None of the cases cited received any form of postoperative treatment.

In general terms I think it may be said that the results of cancer are bad in proportion to the accuracy of the microscopic examinations, and at the New York Hospital we are fortunate in having so recognized an authority as Dr. Elser, director of laboratories, and we also enjoy the coöperation of Professor Ewing, who has often been appealed to for important advice and decisions.

Our ward patients present a very sad picture of having consulted many physicians before any intelligent attempt was made to determine their conditions. Particularly sad are the victims of the gastrointestinal tract—pills, powders, diet, etc., for indigestion without any kind of examination. The cancer of the colon is treated for constipation until acute obstruction occurs; those with bleeding are given pile remedies (?) without any kind of an examination.

Our histories contain a specific query of what happened to the patient the first time he consulted a doctor. With rare exception were they given advice or examination of any value.

The Value of the Palliative Operation. For the laity an operation is an operation. If we yield to the importunities of relatives and perform a gastroenterostomy, gastrostomy or colostomy, as an act of mercy, surgery is credited with a failure to cure.

What is the outlook for improvement, early diagnosis and treatment? I think the public will solve that question rather than the profession. There was much the same problem in tuberculosis, and the decided improvement began when the public, including the poor and ignorant, learned for themselves what the problem was and how to handle it.

We have been living in a fool's paradise of fallacious statistics. All the older figures should be ruthlessly junked and so-called

radical operations should only be performed after the most painstaking search for metastases are exhausted. Outside of visible and palpable manifestations, we have nothing but *x*-ray examinations for skeletal or intrathoracic deposits and these demonstrable only after attaining some size. A really comprehensive *x*-ray examination means examination of practically the whole body and infinite labor and expense.

The investigation for possible metastases is shown in the two tables. While of late we are making a creditable showing, *e. g.*, 93 per cent examination in breast cases, it is a most mortifying lesson of our slowness in realizing the importance of this development. On the whole, the positive evidence obtained has not been very great, but Dr. Wade, of our house staff, will later publish a more detailed analysis of the material, which we trust will be helpful.

TABLE VIII.—SUMMARY OF *X*-RAY EXAMINATIONS FOR METASTASES IN CASES OF CARCINOMA ADMITTED TO THE FIRST SURGICAL DIVISION OF THE NEW YORK HOSPITAL, 1914–1925

Group.	Cases.
I. Carcinoma of gall-bladder and ducts, liver, esophagus, pancreas and stomach	220
II Carcinoma of intestine, mesentery, omentum, peritoneum, rectum and peritoneal glands	82
III. Carcinoma of breast	86
IV. Carcinoma of female reproductive organs	65
V. Carcinoma of other organs	60

Approximately 75 patients have received by our direction some form of radiotherapy, either postoperatively or when radical operation could not be performed. The postoperative radiation was used chiefly in the bad cases, where the operative outlook was poor. Most of the radiotherapy was given at the Memorial Hospital, to which we are greatly indebted for its great assistance. As regards helpful results, we can give no definite statement. We regret to record, however, that our personal impression is that no real improvement except moral effect of doing something has been attained by radiotherapy.

We realize that occasionally brilliant reports are circulated, but believe that these belong mainly to the class of patients we have cited as surviving many years despite obvious lesions.

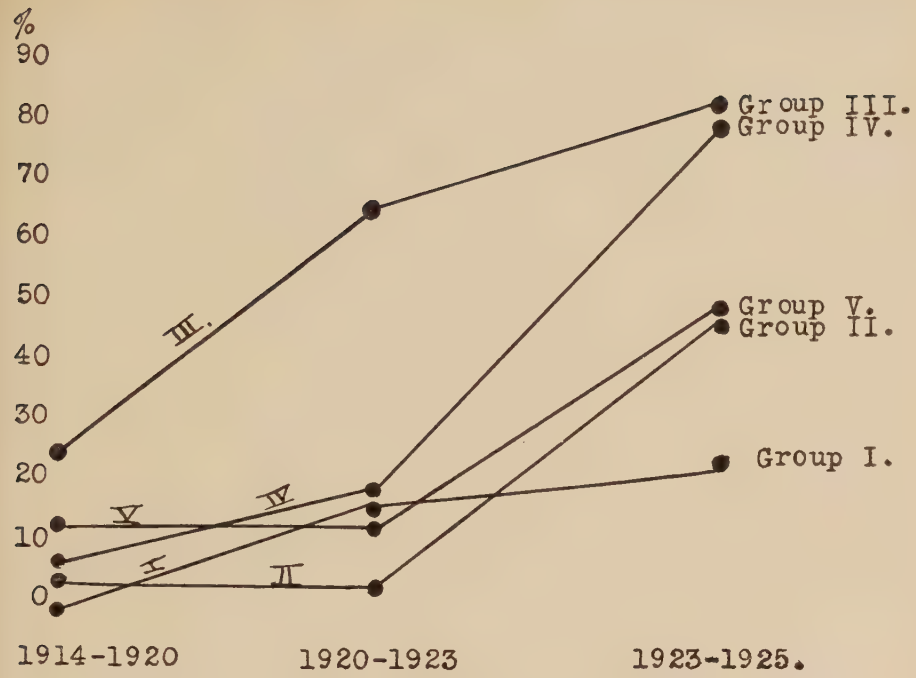

CHART I.—Curve showing percentage of *x*-ray examinations of carcinoma patients with metastases.

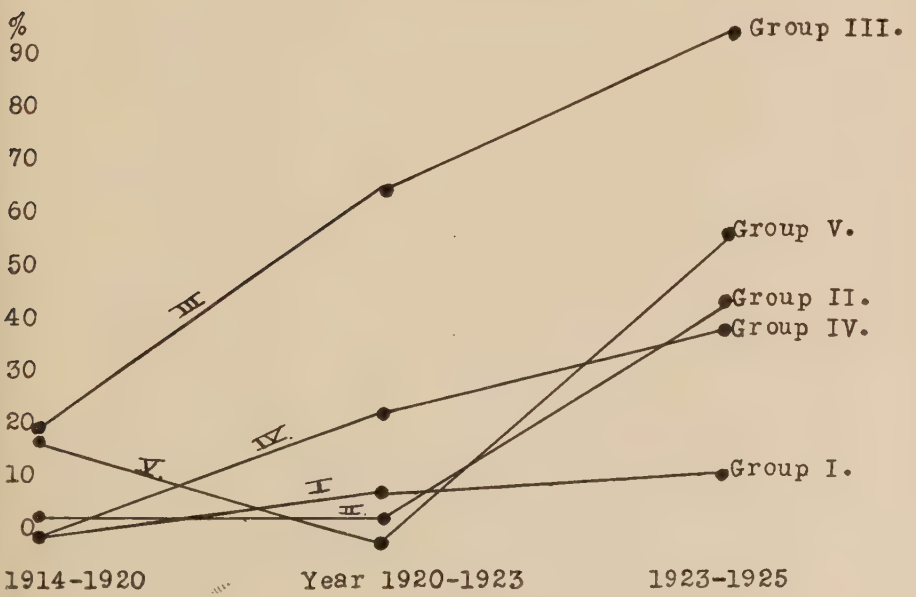

CHART II.—Curve showing percentage of *x*-ray examinations taken before operation on carcinoma.

Radiotherapy is often attended with disastrous complications which add to the misery of these unfortunates. The natural anxiety of relatives to "do something" often leads also to pitiful impoverishment.

Summary of Malignant Tumors

(*Explanation of symbols:* * died following operations; † *x*-ray or radium treatment; ‡ never seen after discharge.)

Carcinoma of Appendix. 7 operations; 4 living, 1 unknown.

Radical operation, all.

1.*

1 died 3 years 9 months after discharge; recurrence in 6 months.†

1 unknown.‡

4 living:

5 years postoperative (History Nos. 198728 and 226443).

4 years postoperative (History Nos. 238173 and 236015).

Carcinoma of Breast. 76 operations; 17 living, 7 unknown.

Radical operation, 75.

1 case partial excision only, as case was clearly hopeless.

1.*

50 died after discharge (20†).

1 died 11 years after operation, at the age of 70 years, of apoplexy; breast condition absolutely all right (191293).

Living cases. No recurrence.

10 years 6 months (199164)

8 years 4 months† (210519).

6 years† (226531).

5 years (232135).

4 years 6 months (200460, 237187).

3 years 5 months (241982).

2 years (252008,† 248946†).

1 year (258406, 257360,† 255532†).

9 months (252137†).

Living cases. Recurrences.

9 years postoperative; site of operation O.K.; operation for carcinoma of opposite breast (263577).

3 years postoperative; living 9 months later (239517).

1 year postoperative;† living 4½ years later (229198).

4 years postoperative;† living 3 years later, but condition very poor (220740).

Carcinoma of Face. 6 operations; 1 death, 4 living, 1 unknown.

Radical operation, excision of tumor, in all cases.

1 died 2 months postoperative; at time of operation metastatic lymph nodes removed.†

Living cases. No recurrence.
6 years (192637).
5 years (203160).
2 years 10 months (243674).
10 months (252345).
Unknown, 1. O.K. 11 months postoperative; never seen again

Carcinoma of Gall-bladder. 15 operations; 15 deaths.
Cholecystostomy, 1; died 2 months later.
Cholecystotomy, 1.*
Cholecystenterostomy, 1; died 5 months postoperative.
Cholecystectomy, 3.
2.*
1 died 5 months postoperative.
Cholecystocolostomy, 1.*
Exploratory, 7.
2.*
3 died 1 month postoperative.
1 died 3 months postoperative.
1 died 4 months postoperative.
Exploratory and nephrectomy, 1.*
NOTE: All patients dead within 6 months.

Carcinoma of Intestines. 50 operations; 41 deaths, 3 living, 6 unknown.
Exploratory, 19; all known cases are dead.
8.*
6 died within 6 months.
1 case had first- and second-stage Mikulicz for diverticulitis 10 months previously; died 3 months after second operation.
1 died 2½ years postoperative; definite metastases in liver 7 months postoperative.
Unknown, 3.
2.‡
1 followed 5 months; at that time losing weight and strength rapidly.
Radical operation, 31.
18.*
1 metastasis, 2 months; dead 4 months postoperative.
1 died 1 month postoperative.
1 died 6 months postoperative.
1 metastasis 1 year; dead 4 months later.
1 metastasis 3 years 8 months; dead 1½ years later.
1 metastasis 1 year; dead 2 years postoperative.
1 died following operation in Greece 2½ years after operation here; nothing known about metastasis.

Appearance of metastases.
13 within 6 months.
11 within 1 year.
13 within 2 years.
7 within 3 years.
4 within 4 years.
3 within 5 years.
2 within 6 years.
1 within 9 years.
Unknown, 7.
1;‡ 3 months O.K.; 4 months, O.K.; 6 months, O.K.; 9 months (1 O.K.; 1 recurrence); 3 years, O.K.

Carcinoma of Bartholin's Gland. 1 operation; 1 death.
Radical operation, 1; metastasis within 4 months; dead 8 months postoperative.

Carcinoma of Bladder. 8 operations; 7 deaths, 1 unknown.
Excision of tumor, 3.
1.*
1 died 2 months postoperative.
1 died 5¼ years after discharge of a condition in no way related to her carcinoma; never had a recurrence; she was in her 73d year at the time of her death (217091).
Exploratory, 1.*
Cystostomy, 4.
1.*
1 died 8 months postoperative.
1 died following second operation 1 year later.†
1 unknown; followed 16 months; unimproved.

Carcinoma of Buccal Mucous Membrane. 1 operation; 1 unknown.
Excision by cautery, 1. 3 months postoperative all right; never seen again.

Carcinoma of Broad Ligaments. 1 operation; 1 unknown.
Excision of tumor, 1.‡

Carcinoma of Esophagus. 4 operations; 3 dead, 1 unknown.
Gastrostomy, 4.
2.*
1 died 1 month postoperative.
1 distinctly weaker 1 month after discharge; never seen again.
Living, 3. No recurrence, 2.
7 years (223299); has already been quoted as living with no complaints, notwithstanding metastases found at operation.
3 years 7 months (238479).
Recurrence, 1.
Recurrence in uterus one year later; 6 months later still living but condition hopeless (256805).
Unknown, 3.
1‡.
1 recurrence one year later; never seen again.
1 living two and a half years after operation; never seen again.

Carcinoma of Kidney. 2 operations; 1 living, 1 dead.

Nephrectomy, 2.

1 metastases to brain 3 years 7 months postoperative; died 1 month later.

1 living 2 years 2 months postoperative; O.K.

Carcinoma of Larynx. 2 operations; 2 deaths.

Tracheotomy, 1;† radium treatment following operation; 1 year after operation, report from Memorial Hospital: "Process certainly at a standstill; 3 examinations show absence of carcinoma;" died 1 year 7 months later.

Gastrostomy, 1.; died 2 months later.

Carcinoma of Leg. 1 operation; 1 death.

Amputation below the knee; metastases in brain 3 months postoperative; died 5 months later.

Carcinoma of Lip. 10 operations; 3 deaths, 2 living 5 unknown.

Excision of tumor, 10.

1 huge recurrence 1 year 2 months after discharge; died 3 months later.

1† recurrence 10 months postoperative; came in for removal of nodes under chin; radium treatment of no avail; died 4 months later.

1 died 2 years postoperative; date of metastases not known.

Living cases. No recurrence.

2 O.K. 5 years postoperative (194357, 204681).

Unknown.

2.‡

1 O.K. 2½ years postoperative; never seen again.

1 came in 3 months after discharge for removal of cervical nodes, a prophylactic measure; microscopic examination showed no evidence of metastasis; patient never seen again.

1 O.K. 6 months after discharge; never seen again.

Carcinoma of Liver. 11 cases, all exploratory; all dead.

Carcinoma of Lymph Nodes. 7 operations; 5 deaths, 2 unknown.

Carcinoma of Maxilla. 2 operations; 1 death, 1 unknown.

Carcinoma of Nose. Cauterization of tumor;† died 9 months later.

Carcinoma of Omentum. 2 operations; 2 deaths.

Exploratory, 2; 1 died 2 weeks after leaving hospital; the other 4 months.

Carcinoma of Pancreas. 4 operations; 4 deaths.

Cholecystostomy, 2.*

Exploratory, 2.*

Carcinoma of Penis. 1 operation; 1 living; no recurrence 1 year later.

Carcinoma of Peritoneum. 3 operations; 3 deaths.

Exploratory, 5.*

Carcinoma of Prostate. 3 operations; 2 deaths, 1 unknown.

Prostatectomy, 3.

1.*

1 recurrence 10 months postoperative; died 7 months later.

1.‡

Carcinoma of Rectum. 12 operations; 10 deaths, 1 living, 1 unknown.

Colostomies, 6.

1 living 3 years postoperative† (242633); others all dead or hopeless.

Resections, 3; all dead.

Exploratory, 3; all dead.

Carcinoma of Retroperitoneal Nodes. 1 operation; 1 death.*

Carcinoma of Stomach. 69 operations; 4 living, 6 unknown.

Exploratory, 32.

6.*

With exception of 4 unknown, all remaining cases died within 6 months.

Jejunostomy, 4.

3.*

1 died 2 months after discharge.

Gastroenterostomy, 18.

7.*

5 died within 6 months.

2 died within 1 year.

1 had local deposits in scar 10 months after discharge; died 1 year later.

1 case large mass 11 months postoperative; still living, 1 year postoperative.

1 unknown, O.K. 10 months postoperative.

1 died 18 months postoperative.

Resections, 15.

8.*

1 died 1 month after discharge.

1, metastasis a little over 1 year; died 1 year 7 months postoperative.

1, metastases 1 year later; died 1 year 3 months postoperative.

Living. No recurrence.

3 years 8 months O.K. (240504).

4 years 2 months O.K. (237171).

Living. Recurrence.

3½ years postoperative; still living, 3 years 9 months postoperative (242284).

Unknown, 1.‡

Carcinoma of Scrotum. 1 operation; 1 living.

Excision, 1; no recurrence 5 years 11 months postoperative (226595).

Carcinoma of Skin of Shoulder. 1 operation; 1 death.

Excision, 1; recurrence 2 months postoperative; died within 1 year.†

Carcinoma of Submaxillary Gland. 1 operation; 1 living.

Removal of tumor, 1; O.K. 2 years 2 months postoperative (249662).

Carcinoma of Testicle. 1 operation; 1 death.

Castration, 1; dead 1 year 4 months later.

Carcinoma of Thumb. 1 operation; 1 unknown.

Amputation of thumb, 1; O.K. 4 months postoperative; never seen again.

Carcinoma of Thyroid. 1 operation; 1 living.

Excision, 1; O.K. 4 years 5 months postoperative (235795).

Carcinoma of Tongue. 7 operations; 1 living, 6 dead.

Excision.

1 died 2 months postoperative.

1† recurrence 1 month after discharge; died 9 months later.

1† recurrence 1 year 4 months postoperative; died 1 year 8 months postoperative.

1 recurrence 9 months postoperative; died 14 months postoperative.

1 enormous recurrence, nodes of neck, 3 years postoperative; died 3 years 3 months postoperative.

1† recurrence 2 months postoperative; died 11 months postoperative.

Living, 1; discharged cured 10 years postoperative (196548).

Carcinoma of Tubes and Ovaries. 13 operations; 10 deaths, 1 living, 2 unknown.

Radical.

1.*

1† died 3 months postoperative.

2 died 3 months postoperative (1 died of influenza).

1 died 11 months postoperative; massive recurrence.

1 died 1 year 5 months after discharge.

1 died 2½ years after discharge.

2 unknown (1 O.K. 3 months after discharge).

Living, 1; O.K. 3 years 5 months postoperative (244421).

Exploratory.

1† died 5 months postoperative.

1.*

1 died 1 year 8 months postoperative.

Carcinoma of Uterus.

Hysterectomy, 22.

4.*

1 died 6 months later.

2 recurrences within 6 months; dead 10 months postoperative (1 died 1 year postoperative).

1 recurrence 1 year later;† second operation 2 years after first operation; died 2 years later.

1 metastasis 2 years 3 months; died 1 year 10 months later.

1 metastasis 1 year 6 months; died 1 month later.

1 recurrence 2 years vaginal vault; excision; another recurrence 2 years later and died following second operation.

1 recurrence 2 years;† died 1 year later.

1 hysterectomy for fibroids 1921; biopsy and cauterization of cervix for carcinoma 3½ years later;† recurrence 1 year later; died 1 year 4 months after second operation.

1 recurrence 2 months after discharge;† died 11 months postoperative.

1 recurrence 3 months; died 1 year later.

1 recurrence 7 months; died following treatment with radium 2 years 3 months postoperative.†

Living cases:

4 years 9 months postoperative, O.K. (231528).

7 years 7 months postoperative, O.K. (216782).

2 years 9 months postoperative, O.K. (246283).

Unknown.

1 patient went to Italy seven months postoperative; no recurrence at that time; never heard from again.

Cauterization, 6.

2 died one and a half years later (1†).

1 died nine months postoperative.

1 died 1 year later.

2.‡

Exploratory, 4.

1† progressively worse; died 9 months postoperative.

1 died 2 months postoperative.

1 died 5 months postoperative.

1 died 1½ years postoperative.

Dilatation and curettage; biopsy, 1.

1† bad condition 2 years 3 months postoperative (249548).

Dilatation and curettage, trachelorrhaphy, shortening of round ligaments, 1.

1† good condition 3 years 4 months later (240503).

Anterior colporrhaphy, perineorrhaphy, excision of tissue for diagnosis, 1.

1† dead 1½ years later.

Closure of vesicovaginal fistula, 1.

1 dead 2 months later.

Excision of tumor, 1.

1† followed by hysterectomy; O.K. 2½ years postoperative (245830).

Carcinoma of Vulva. 1 operation; 1 death.

Excision, 1; marked recurrence 3 months; died 6 months postoperative.

Endothelioma of Arm. 1 operation; 1 death.

Excision of tumor, 1; died 7 months postoperative.

Endothelioma of Face. 1 operation; 1 unknown.

Excision, 1;† unimproved 1 year 10 months postoperative; never seen again.

Endothelioma of Lymph Nodes. 7 operations; 3 living 4 unknown.

Excision, 7.

Living, 3.

2½ years postoperative (239660).

6 years postoperative (198479).

1 case excision of axillary lymph nodes;† recurrence in cervical lymph nodes 4 years 7 months after first operation; second excision;† O.K. 1 year 8 months after second operation (224496).

Unknown, 4.

1.‡

1 extensive recurrence 3 months postoperative; end-result not known.

2 cases O.K. 7 months after discharge; no further trace.

Endothelioma of Neck. 1 operation; 1 living.

Excision, 1; O.K. 9 months postoperative (195594).

Endothelioma of Ovary. 1 operation; 1 living.

Excision, 1; O.K. 3 years postoperative (207492).

Endothelioma of Parotid. 4 operations; 2 living 2 unknown.

Excision, 4.

Living. No recurrence.
3 years 9 months (239510).
4½ years (196858).
Unknown, 2.
1.‡
1 O.K. 3 months; not seen again.

Sarcoma of Abdominal Wall. 1 operation; 1 death.
Excision of tumor, 1; died 4 years postoperative.

Sarcoma of Axilla. 1 operation; 1 death.
Excision of tumor mass, 1; patient died 4 months later.

Sarcoma of Bones. 13 operations; 6 deaths, 7 living.
Radical excision or amputation, 12.
1.*
3 died within 6 months.
1 recurrence 1 year 8 months postoperative;† died 2 years 4 months postoperative.
Living.
2 years 2 months (249667); femur.
3 years 1 month (223451); ulna.
4 years 10 months (233356†); tibia.
5 years 7 months (228446); radius.
6 years (221900);† humerus.
8 years 7 months (206054); femur.
10 years (194540); humerus.
Incision and drainage and biopsy, 1.*

Sarcoma of Breast. 2 operations; 2 deaths.
Amputation, 2.
1 had second operation 2 weeks after first for new growth of abdomen.*
1 recurrence 6 months; dead 11 months postoperative.

Sarcoma of Gums. 1 operation; 1 living.
Excision, 1; so far as sarcoma goes, O.K. 10 years postoperative; has an old pulmonary tuberculosis.

Sarcoma of Groin. 1 operation; 1 death.
Excision, 1; metastases in spine 3 months postoperative; died within 9 months postoperative.

Sarcoma of Intestines. 10 operations; 7 dead, 2 living, 1 unknown.
Resections, 5.
1.*
1 died 1 week after discharge.
1 died 3 years 7 months later.
1 living; O.K. 8½ years postoperative (211240).
1‡ unknown.
Ileostomy, 1.*
Exploratory, 4.
1 died in 1 month.
2 died in 4 months.
1 still living, 2 years postoperative; bad condition (251419).

Sarcoma of Kidney. 2 operations; 2 deaths.
Nephrectomy, 2.
1 died 1 month postoperative.
1 died 6 months postoperative.
Sarcoma of Lymph Nodes. 3 operations; 1 living, 2 unknown.
Excision, 2.
1 living; O.K.† 5 years 9 months postoperative (228666).
1 unknown; had a recurrence 1 year 3 months postoperative.
Exploratory, 1.
No specimen; O.K. 10 months postoperative; went to Italy and not heard from again.
Sarcoma of Neck. 1 operation; 1 living.
Excision of tumor, 1; O.K. 5¼ years postoperative (228987).
Sarcoma of Ovary. 2 operations; 2 deaths.
Excision, 2.
1 died 10 months postoperative.
1 recurrence 11 years postoperative; died 7 months later.
Sarcoma of Spleen. 2 operations; 2 deaths.
Splenectomy, 2.
1.*
1 died 3 months postoperative.
Sarcoma of Stomach. 2 operations; 1 death, 1 living.
Resection, 2.
1 recurrence 10 months postoperative;† died 1 year 5 months postoperative.
1 living; no recurrence 5½ years postoperative (229363).
Sarcoma of Rectum. 1 operation; 1 unknown.
Excision, 1.‡
Sarcoma of Testicle. 1 operation; 1 death.
Castration, 1; recurrence 1 year 7 months;† died 3 months later.
Sarcoma of Tendon Sheath. 1 operation; 1 living.
Removal of tumor, 1; O.K. 2 years 3 months postoperative (214561).
Sarcoma of Uterus. 1 operation; 1 living.
Hysterectomy, 1; O.K. 5 years 7 months postoperative (230854).
Sarcoma of Upper and Lower Extremities (Not Bones). 9 operations; 3 deaths, 3 living, 3 unknown.
Radical excision, 8.
1† had second operation 10 months after first operation; died 1 year 2 months later.
1 died 1 year 5 months postoperative.
2 unknown (1 at end of 3 months was not well).
3 living; no recurrence.
2 years 3 months postoperative (210187).
2 years 9 months postoperative (246458).
3½ years (241185).
Exploration, 1; aspiration and biopsy of tumor of thigh; died 2 months postoperative.

Hypernephroma and Teratoma.

Kidney. 2 operations; 2 deaths.

Nephrectomy, 2.

1 died 1 month after discharge.

1 recurrence in 11 months;† died 1½ years postoperative.

Retroperitoneal Nodes. 1 operation; 1 death.

Exploratory, 1; died 3 months postoperative; primary growth in testicle.

Testicle. 1 operation; 1 death.

Orchidectomy;† metastases in lungs 5 months postoperative; died 3 months later.

EXOPHTHALMIC GOITER AND TOXIC ADENOMA: SIMILARITY OF RESPONSE TO IODINE* †

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Recent American surgical literature conveys the impression that exophthalmic goiter and toxic adenoma are two different diseases. The differentiation is said to depend upon certain distinctive clinical, pathological and therapeutic observations. We have been led to question seriously the validity of this conception because our own experience has not confirmed such differential findings.

We have discussed previously[1] the clinical manifestations and anatomical and histological alterations in the thyroid in exophthalmic goiter and "toxic adenoma." Any clinical and pathological differences we have been able to recognize have not appeared to be of such fundamental significance as to establish these two clinical states as distinct diseases. These aspects of the subject will not be considered further at this time.

The purpose of the present paper is to point out the similarity of the response to iodine, as a measure preliminary to operation, in a series of cases of exophthalmic goiter and "toxic adenoma," selected in accordance with certain definite requirements for comparison.

Although it is well established that iodine is distinctly beneficial as a preoperative preparation for thyroidectomy in cases of exoph-

* Read by title.

† From the Department of Surgery, The Lakeside Hospital and The Western Reserve University School of Medicine.

thalmic goiter, its use in any manner whatever in cases of "toxic adenoma" has been specifically condemned.[2]

The following impressions, gained from current literature, seem to reflect present-day views concerning this subject, and are set forth here as matters of interest in connection with observations we have to present:

1. Iodine is being used as a preoperative preparation in surgical clinics generally in cases of exophthalmic goiter but not in cases of toxic adenoma.

2. Exophthalmic goiter seems to be regarded as an essential dysthyroidism in the sense of an intoxication resulting from excessive quantities of perverted or incompletely iodized thyroid secretion.

3. Toxic adenoma seems to be regarded as an essential hyperthyroidism in the sense of an intoxication resulting from excessive quantities of normal or completely iodized thyroid secretion.

4. There appears to be a dilemma in certain cases that are said to have both exophthalmic goiter (dysthyroidism) and toxic adenoma (hyperthyroidism) at the same time. Iodine is known to be beneficial as a preoperative preparation in the former but is said to be contraindicated in the latter.

5. Of particular interest and possible significance are certain cases in which clinicians of experience have made a definite clinical diagnosis of toxic adenoma, have withheld iodine before operation in view of that diagnosis, and have been astonished to observe a "typical postoperative hyperthyroid reaction," and even death, following thyroidectomy. The unexpected postoperative reaction, or death, seems to have been accepted as a clear indication of mistaken preoperative clinical diagnosis which was then changed accordingly from toxic adenoma to exophthalmic goiter. The change of diagnosis seems to have been supported further by the finding of certain histological alterations in the thyroid supposed to be characteristic for exophthalmic goiter (a supposition we regard as not well founded). The failure to utilize iodine as a preparation for thyroidectomy has been a source of chagrin in such cases.

6. The postoperative mortality is higher in cases of toxic adenoma than in cases of exophthalmic goiter.

7. There appears to be a third disease, "iodine hyperthyroidism,"[3] which is neither exophthalmic goiter nor toxic adenoma.

(With this disease the writers have had no personal experience. At all events it has no place in the present discussion since it is neither exophthalmic goiter nor toxic adenoma.)

Material. In a series of 50 consecutive admissions to the general surgical division of The Lakeside Hospital there are 30 cases of toxic goiter and 20 cases of nontoxic goiter. Among the 30 cases of toxic goiter there are 20 of the exophthalmic type and 10 which correspond to what is called toxic adenoma.

For the purpose of comparing the response to iodine as a preoperative preparation for thyroidectomy in cases of exophthalmic goiter and toxic adenoma, we have selected from this series all cases that conformed to the following standard requirements:

1. Primary admissions to the general surgical service without previous operative intervention, iodine treatment, *x*-ray or other definite therapeutic regimen.

2. Two or more preoperative and one or more postoperative determinations of basal metabolic rate.

3. The administration of iodine (Lugol's solution) before operation.

4. Primary thyroidectomy following the administration of iodine.

5. Uncomplicated by active infectious disease, notably tuberculosis.

In accordance with these requirements we have for comparison 10 cases of exophthalmic goiter and 4 cases of toxic adenoma (Charts III, IV, V, VI). It is hoped that this rigorous selection permits us to deal more accurately with the question at hand, than is often the case. Cases not selected for comparison of the two groups as specified may be utilized for the purpose of further illustrating the reaction to iodine under variable conditions of dosage, time relations, influence of pevious therapy, state of the thyroid, and hospitalization (Charts I, II and Table 1).

Concerning the differentiation of the two special groups compared the following may be stated:

1. *Exophthalmic Goiter.* Exophthalmos was present in most cases. There was diffuse parenchymatous hypertrophy and hyperplasia of the thyroid. The thyroid was not adenomatous (clinically), although small adenomata were recognized, after removal, in a few glands. Thrill and bruit were present. Nervous and vasomotor phenomena were prominent features. Tachycardia,

palpitation, increased appetite, loss of weight and strength were well-marked symptoms.

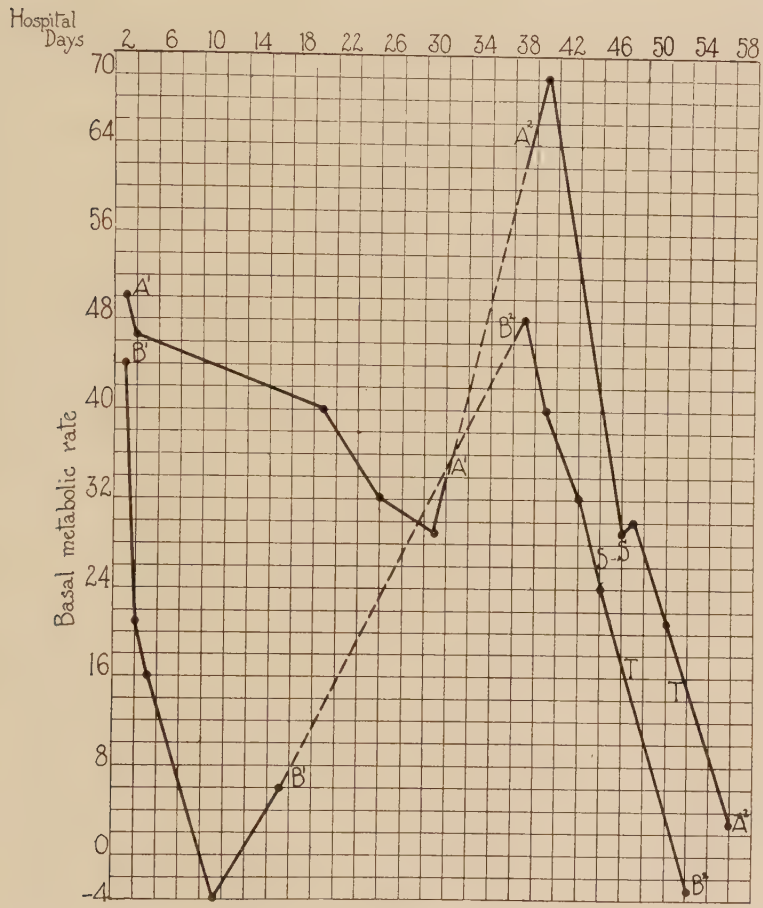

CHART I.——— Hospitalization; ----- Interval between admissions; S, Transfer from medical service; T, Thyroidectomy. Basal metabolic-rate curves (A^1A^2 and B^1B^2) of 2 cases of exophthalmic goiter. Observation period is about two months, during which time there were two admissions to the medical service with final transfer to surgery and primary subtotal thyroidectomy. Case A had no previous treatment. Case B had a previous medical admission of thirty-nine days during which time, with rest and luminal without iodine, the basal metabolic-rate diminished from 41 per cent above normal to 3 per cent below normal. During the succeeding eight months of observation in the out-patient department a series of eight *x*-ray treatments but no iodine was given. Further details of these 2 cases are shown in the first two columns of Table I.

2. *Toxic Adenoma.* Exophthalmos was absent in all cases. Adenomatous goiter was the outstanding clinical feature. Nothing comparable to the diffuse parenchymatous hypertrophy and hyperplasia that are said to characterize exophthalmic goiter were present. There was no thrill or bruit. Nervousness, vasomotor disturbances, increased appetite, loss of weight and strength, while present in mild degree and in various combinations, in some cases, were not striking clinical features.

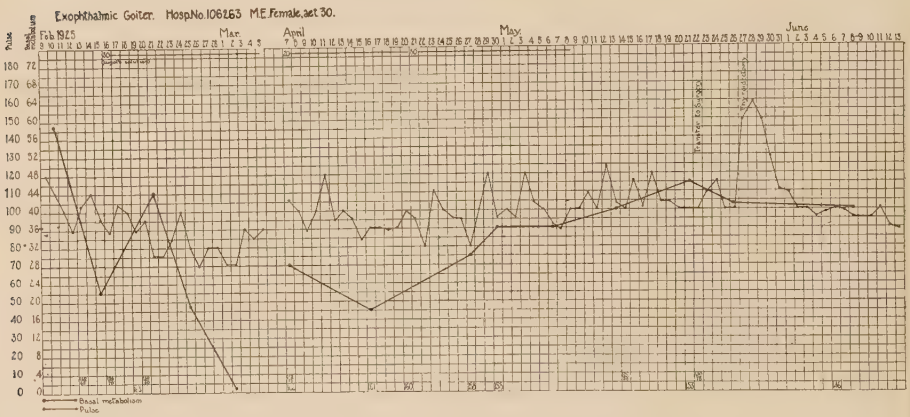

CHART II.—Basal metabolic-rate curve of a case of exophthalmic goiter (Case C) observed over a period of about four months, during which time there were two admissions to the medical service with final transfer to surgery and primary subtotal thyroidectomy. The interval between the two medical admissions was thirty-two days. The medical treatment consisted of rest, luminal and Lugol's solution during both admissions. Note the difference in response when iodine was administered for the first time as compared with its use subsequently. During the second admission there was a slight primary improvement followed by a well-defined secondary aggravation due to the prolonged use of iodine. Further details of this case are shown in the third column of Table I.

EXOPHTHALMIC GOITER. The administration of iodine as a measure preliminary to thyroidectomy in cases of exophthalmic goiter is now a well-established procedure. Our experience is in accord with this principle. The following factors appear to be of importance in determining the degree of clinical response:

1. The age and physical state of the patient.
2. The intensity and duration of the disease.
3. The physical and chemical state of the thyroid gland at the beginning of iodine therapy.
4. The degree of elevation of basal metabolic rate above normal.

5. Whether or not the patients previously have been taking iodine.

6. The presence of complicating conditions—tuberculosis, pyogenic infection, pregnancy, diabetes, nephritis, cardiac disease, etc.

The quantity of iodine necessary to accomplish the maximum clinical benefit previous to operation depends upon the combination of factors outlined. Patients that have had iodine before coming to the surgeon show great variations in the degree of response to preoperative iodine, and equally great variations in the quantity of iodine necessary to bring about a condition in which operation is safe. Under such circumstances the surgeon may be deprived of practically all the immediate advantages of preoperative iodine and is confronted with the alternatives of performing a hazardous thyroidectomy, or resorting to procedures of lesser magnitude such as ligations or hemithyroidectomy, or of waiting for a period before reinstituting complete iodine therapy. Fortunately such cases constitute only a small percentage of the total and are practically limited to those that have been taking iodine in large quantities or for a prolonged period of time immediately before admission to the surgical clinic. In such cases iodine should not be discontinued entirely (Chart II), but thyroidectomy should be performed as early as possible or a lesser procedure resorted to in accordance with the clinical judgment of the surgeon.

Cases in which there has been an interruption of the previous iodine therapy are usually amenable to preoperative iodine (Chart I) particularly if the interval without iodine has been three to four weeks or longer. When this free interval amounts to three months or more, the previous iodine therapy may be disregarded so far as the resumption of iodine as a preoperative measure is concerned.

In those hospitals where patients with exophthalmic goiter are admitted to the medical service and are to be transferred to surgery for operation, iodine therapy may be instituted on the medical service but the surgeon should have the opportunity of selecting the time for operation and the type of operative procedure. With a proper coöperation on the part of the two services neither the patient, the internist, nor the surgeon need necessarily be at any particular disadvantage, and in some respects the patient's ends may be better served.

TABLE I

	Chart I.		Chart II.
	Case A.	Case B.	Case C.
First admission (medical):			
Days in hospital	31	16	25
Basal metabolic rate:			
Admission	+50%	+44%	+59%
Discharge	+29%	+ 6%	+ 1%
Iodine (Lugol's solution):			
Hospital day started	15	1	8
Number of days given	17	16	18
Minims per day	15	15	30
Total quantity (minims)	255	240	540
Interval between admissions:			
Days	6	21	32
Iodine	none	none	none
Second admission (medical):			
Basal metabolic rate:			
Admission	+70%	+48%	+28%
On transfer to surgery	+29%	+24%	+46%
Preoperative	+29%	+24%	+40%
Postoperative	+ 3%	− 3%	+40%*
Iodine (Lugol's solution) preoperative:			
Hospital day started (medical service)	5	4	2
Number of days given (medical service)	6	5	31
Minims per day	45	10	20 to 30
Total quantity (minims) (medical service)	270	50	800†
Total quantity (surgical service)	225	30	none‡
Postoperative reaction	slight	slight	marked
Hospitalization (days):			
Iodine started (medical service)	5	4	2
Transfer to surgical service	10	9	46
Hospital day of operation	14	12	51
Surgical day of operation	5	4	6
Total hospital days (medical and surgical)	24	24	68

* See legend for explanation.
† Discontinued on the medical service fourteen days before transfer.
‡ Not resumed on the surgical service.

TABLE I.—Details of 3 cases (A, B, and C), of exophthalmic goiter, the basal metabolic rate curves of which are shown in Charts I and II, that have had two or more previous admissions to the medical service before transfer to surgery. Lugol's solution was administered during the medical hospitalization but not during the intervals between admissions.

The following points of interest are illustrated:

1. The failure of iodine to cure exophthalmic goiter even though the basal metabolic rate decrease to within normal limits after variable quantities of the drug for variable lengths of time.

2. The more favorable response to iodine when given for the first time as compared with the response to its use subsequently. (Compare Chart III with Charts I and II.)

3. Of 2 cases receiving the same quantity of iodine per day and for about the same number of days, a greater percentage reduction of basal metabolic rate occurred in

There is little doubt that from the standpoint of the surgeon the most acceptable cases of exophthalmic goiter are those that come under observation without previous treatment of any kind (Chart III). In such cases a carefully planned preoperative preparation, including the administration of iodine, permits a primary thyroidectomy in a very high percentage of cases and is followed by a minimum of postoperative reactions. Although the hospitalization is somewhat increased by reason of the iodine therapy, repeated admissions are minimized, incomplete and multiple-stage operations are largely done away with, the maximum surgical therapy is accomplished in a shorter period of time, and the patients have the advantage of earlier restoration of their economic status.

Toxic Adenoma. Within the past few years "toxic adenoma" as an entity, in contradistinction to exophthalmic goiter, seems to have met with increasing acceptance. The basis for differentiation depends in no small measure upon a supposed directly opposite response to iodine in the two instances. Indeed the administration of iodine has been proposed as a method of making the distinction in borderline or doubtful cases.[4] Furthermore, the condition called toxic adenoma is said to be brought about by the administration of iodine to borderline toxic cases or to cases of simple endemic goiter with adenomatous thyroids. These tenets are in

the case (Chart I, curve B) in which iodine was instituted on the first hospital day, as compared with the case (Chart I, curve A) in which iodine was instituted on the fifteenth hospital day.

4. On receiving iodine for the second time, after intervals without iodine varying from six to thirty-two days, the 3 cases show a decrease of basal metabolic rate accompanied by a corresponding improvement of clinical manifestations. In 2 cases (Chart I) iodine was given nine and eight days, respectively, on the medical service and was continued on the surgical service to the date of operation, five and four days, respectively, after transfer. The response was as favorable as we usually see in previously untreated cases of equal severity. The postoperative recovery was equally favorable, although the technical difficulties of the operation appear to be increased in such cases as these.

Contrast the foregoing 2 cases with the third case (Chart II) in which iodine was continued thirty-one days (second admission) a total of 800 minims of Lugol's solution being administered. Note the period of primary improvement followed by well-defined secondary rise due to the prolonged use of iodine in large quantities. Iodine was discontinued on the medical service, fourteen days before transfer and was not resumed on the surgical service. This we believe to be a mistake in surgical judgment. There was a severe postoperative reaction. Wound infection and the development of a foul sacral abscess complicated the postoperative course. On discharge the basal metabolic rate was at the same level as before operation. Three months after discharge from the hospital the basal metabolic rate was normal and the patient had gained 40 pounds in weight.

many respects so contrary to our experience that it seems worth while to discuss the matter in some detail.

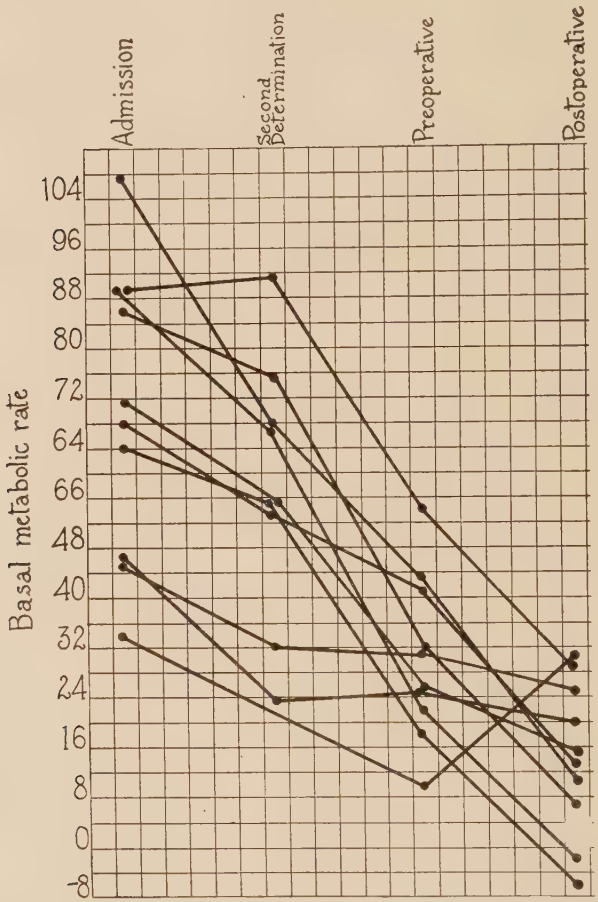

CHART III.—Basal metabolic-rate curves of 10 previously untreated cases of exophthalmic goiter, showing typical response to iodine as a preoperative preparation for thyroidectomy. The basal metabolic rates charted are: The first and second determinations after admission; the last determination before operation; and the average basal metabolic rate after operation, while still in the hospital. Further details are shown in Chart V and Table II.

We know of no symptoms, physical signs, nor alterations in the thyroid which singly are necessarily pathognomonic for the condition called "toxic adenoma," *i. e.*, that are invariably present in

toxic adenoma and invariably absent in exophthalmic goiter or simple endemic goiter.[1]

We recognize fully that patients with adenomatous thyroids and increased basal metabolic rates come to the clinic with the history of having taken iodine as medicine or as iodized table salt, and with the history that their condition has been made worse thereby. From such cases one might readily conclude that iodine caused the disease. Careful inquiry usually discloses the presence of definite symptoms preceding the institution of the iodine. However suggestive these cases may be it is probably not without significance that a history of previous iodine therapy can be obtained in less than one-half of those cases of "toxic adenoma" coming under observation. Moreover, it is to be noted that the same circumstances and sequence of events are frequently encountered in cases of exophthalmic goiter with and without adenomatous thyroids. Our deliberate efforts to produce the clinical state called toxic adenoma by the administration of large quantities of iodine to borderline toxic cases and cases of simple endemic goiter with adenomatous thyroids have been uniformly unsuccessful.

Much emphasis has been placed upon the observation that in cases of established toxic adenoma the clinical condition is aggravated by the administration of iodine. A careful consideration of the reaction to iodine that takes place in cases of exophthalmic goiter, we believe, offers an adequate explanation of the phenomena observed in cases of toxic adenoma under similar circumstances.

As previously noted, cases of exophthalmic goiter that receive iodine for the first time are definitely improved up to a certain point, as indicated by a decrease of basal metabolic rate (Chart III) and amelioration of the clinical symptoms. If iodine be continued in the same dosage for a sufficiently long period of time, the clinical condition becomes aggravated and the basal metabolic rate (Chart II) may increase to its original level or even to a higher level.

In cases of toxic adenoma that come under observation after taking iodine, with a history that the clinical symptoms have been made worse, the question naturally suggests itself: are we not seeing these cases in the period of secondary aggravation that follows a period of primary improvement, similar to that occurring in cases of exophthalmic goiter? In *previously untreated* cases of

toxic adenoma, according to our experience, such a period of primary improvement (Chart IV) following the administration of iodine has been the rule and not the exception.

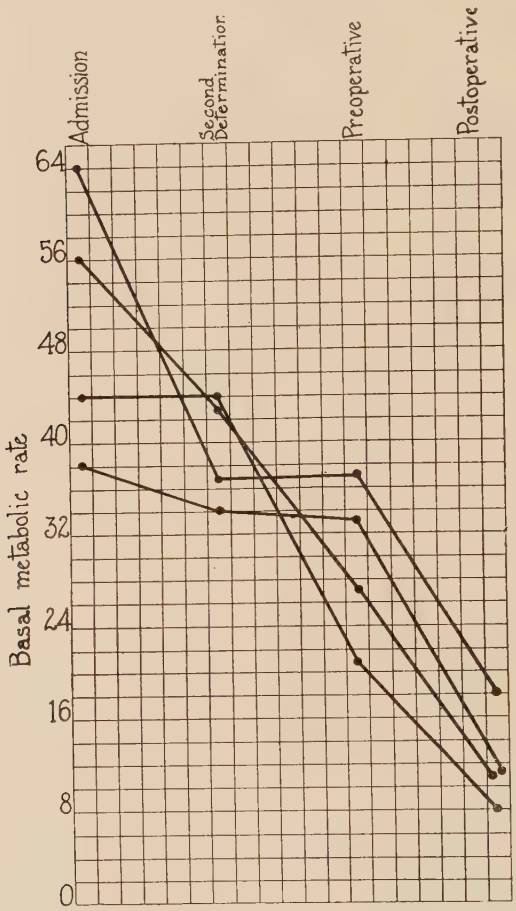

CHART IV.—Basal metabolic-rate curves of 4 previously untreated cases of "toxic adenoma," showing the response to iodine as a preoperative preparation for thyroidectomy. The basal metabolic rates charted are: The first and second determinations after admission; the last determination before operation; and the average basal metabolic rate after operation while still in the hospital. Further details are shown in Chart V and Table II.

CONCERNING DYSTHYROIDISM IN EXOPHTHALMIC GOITER AND HYPERTHYROIDISM IN TOXIC ADENOMA. We have observed that classical cases of exophthalmic goiter, receiving iodine for the first

time, show a period of primary improvement (Charts II, III, VI) followed by a period of secondary aggravation (Charts II, VI), provided the iodine is continued in the same dosage and for a sufficiently long period of time.

We have observed that *previously untreated* cases of toxic adenoma improve in about the same ratio as cases of exophthalmic goiter (Chart V and Table II), following the administration of iodine. They also show a period of secondary increase of basal metabolic rate (Chart VI) when iodine is continued. We have not actually carried out the experiment of continuous iodine therapy in cases of toxic adenoma to the point of obtaining such a degree of secondary aggravation as has been observed by us in cases of exophthalmic goiter following the prolonged use of iodine.

If exophthalmic goiter be a pure form of dysthyroidism—in the sense of an intoxication resulting from an overproduction of a perverted or incompletely iodized product of thryoid secretion—and is greatly improved by the administration of iodine, what explanation do we have for the secondary period of aggravation when iodine is continued? It might be urged that the disease exophthalmic goiter (without adenomatous thyroid) can be converted from a state of dysthyroidism into a state of hyperthyroidism by the use of iodine. If this be so, it would seem logical to expect relief by discontinuing the drug. Experience has shown that the sudden stopping of iodine (Chart II) in cases of exophthalmic goiter that have taken the drug for a long time or in large quantities is rarely followed by any striking clinical improvement and frequently the clinical condition becomes worse. We wish to emphasize that the supposed dysthyroidism, hyperthyroidism and the conversion from one state to the other, in such a hypothetical case occurs in a patient who has not an adenomatous thyroid.

If toxic adenoma be a pure form of hyperthyroidism, in the sense of an intoxication resulting from an overproduction of normal or completely iodized secretion, what explanation have we for the period of primary improvement (Chart IV) noted in *previously untreated cases* in which iodine is administered? It might be urged that during this period of improvement the patient is in a state of dysthyroidism. How can we reconcile the dysthyroidism with the conception that toxic adenoma is a pure form of hyperthyroidism?

Concerning the question of dysthyroidism or hyperthyroidism, and the relation of these states to iodine therapy, the material considered in this paper affords no satisfactory basis for distinction between exophthalmic goiter and toxic adenoma.

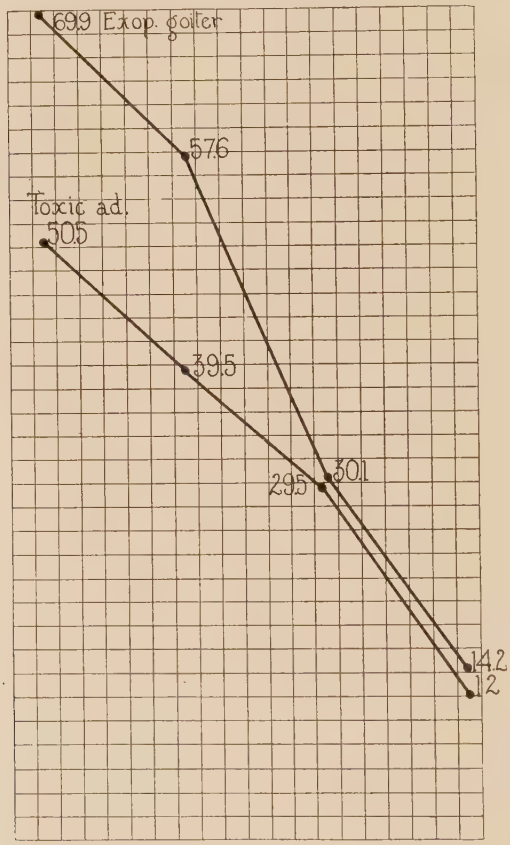

CHART V.—Average basal metabolic-rate curves of 10 cases of exophthalmic goiter and 4 cases of "toxic adenoma." The cases in the two groups are selected in accordance with certain definite requirements for comparison (*vide* text). The metabolic-rates charted are: The first and second determinations after admission; the last determination before operation; and the average basal metabolic-rate after operation, while still in the hospital. The parallelism of the two curves is striking. Further details are shown in Table II.

Our cases of toxic adenoma were not only not aggravated by the administration of iodine but responded in the same manner

and in about the same ratio as did the cases of exophthalmic goiter. *We emphasize again that both groups were previously untreated.*

TABLE II

	Exophthalmic goiter, average (10 cases).	Toxic adenoma, average (4 cases).
Basal metabolic rate:		
Admission	+69.9% (2.4)*	+50.5% (2)
Second	+57.6% (5)	+39.5% (3.25)
Preoperative	+30.1% (18.2)	+29.5% (8.5)
Postoperative	+14.2% (30.7)	+12.0% (18)
Iodine (Lugol's solution):		
Number of days given	16.2	7.5
Minims per day	28.0	22.6
Total quantity, preoperative	452.2	169.5
Hospitalization:		
Day iodine therapy started	4.6	3.5
Day of operation	20.8	11.0
Total hospital days	33.3	19.25

* Numbers in parentheses represent hospital days.

TABLE II.—Details of Charts III, IV and V showing the relations of time (hospital days), daily dosage and quantity of iodine (Lugol's solution) to basal metabolic rate in previously untreated cases of exophthalmic goiter and "toxic adenoma" receiving iodine as a preoperative preparation for primary thyroidectomy.

COMMENT. We appreciate the inadvisability of making generalizations from a small number of observations. However, our experience in this field is more extensive than is represented by the small series of cases here considered. It is proper to state that in the more extended experience we have found nothing fundamentally inconsistent with the observations that form the basis of this paper. In fact, the material considered constitutes an objective confirmation of the views we have previously held.

In this paper we have not been particularly concerned with the question of whether toxic goiter, in its broadest sense, is an essential dysthyroidism, hyperthyroidism or both; whether toxic goiter is a disease in which the thyroid plays a primary or secondary rôle, as regards etiology and pathogenesis.

Our chief concern has been to point out certain inconsistencies in the conception that there are two directly opposed types of toxic goiter (exophthalmic goiter and "toxic adenoma") insofar as any fundamental distinctions between the two supposed types are dependent upon opposite reactions to iodine.

Increased basal metabolic rate and other evidence of "thyrotoxicosis" together with hypertrophy and hyperplasia of the thyroid of any degree, we regard as sufficient indication for the administration of iodine, irrespective of the presence or absence

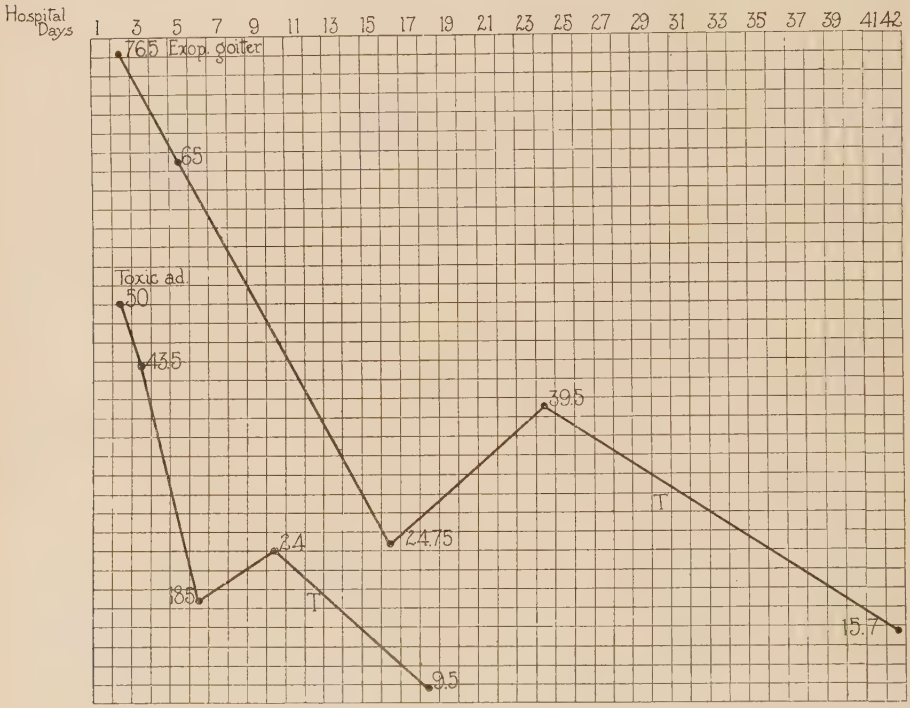

CHART VI.—Average basal metabolic-rate curves of 4 cases of exophthalmic goiter and 2 cases of "toxic adenoma" in which a period of secondary rise succeeds the period of primary improvement following the administration of iodine. All of the cases are taken from the groups represented in Charts III and IV. The basal metabolic rates charted are the following: (1) On admission, (2) at the beginning of iodine therapy, (3) lowest rate during the period of primary improvement, (4) highest rate during the period of secondary rise, (5) the average basal metabolic rate following operation. The two curves are practically identical in character. Time (hospital days) and the quantity of iodine are the variable factors of significance. (See Table II.)

of adenomata. We shall not expect clinical improvement to follow the administration of iodine in any case in which the thyroid is normal or in a completely involuted or colloid state.

It is important to bear in mind that the quantity of iodine

required and the number of days necessary to effect comparable reduction in the basal metabolic rate is far greater in cases of exophthalmic goiter than in cases of "toxic adenoma."

TABLE III

	Exophthalmic goiter, average, (4 cases).	Toxic adenoma, average, (2 cases).
Hospitalization:		
Day of operation	30.0	12.5
Total hospital days	46.25	18.5
Iodine (Lugol's solution):		
Hospital day started	5.5	3.5
Minims per day, period of improvement	25.54	21.0
Total quantity, period of improvement	319.0 (12.5)*	52.5 (2.5)
Total quantity, period of secondary rise	247.5 (7.5)	82.5 (4)
Total quantity, preoperative	713.5 (24.5)	196.5 (9)
Basal metabolic rate:		
Admission	+76.5% (2.75)	+50.0% (2)
At start of iodine	+65.0% (3.5)	+43.5% (3)
Lowest, period of improvement	+24.75% (18)	+18.5% (6)
Highest, period of secondary rise	+39.5% (25.5)	+24.0% (10)
Postoperative	+15.7% (42)	+ 9.5% (18)
Reduction of basal metabolic rates, period of improvement:		
Per cent	61.9	57.5
Days required	14.5	3.0
Lugol's required, minims	319.0	52.5
Increase of basal metabolic rate, period of secondary rise:		
Per cent	55.5	30.0
Days required	7.5	4.0
Lugol's required, minims	247.5	82.5
Ratio of basal metabolic rates:		
On admission	100.0	65.4
At start of iodine therapy	100.0	66.9
Lowest, period of improvement	100.0	74.7
Highest, period of secondary rise	100.0	60.7
Ratio of maximum reduction of basal metabolic rate during period of primary improvement:		
Lugol's required	6.0	1.0
Days required	5.0	1.0

* Numbers in parentheses represent hospital days.

TABLE III.—Details of 4 cases of exophthalmic goiter and 2 cases of "toxic adenoma," the average basal metabolic-rate curves of which are shown in Chart VI. In these cases a period of primary improvement and a period of secondary rise, following the administration of iodine, are illustrated. Although the response to iodine is of the same character in cases of exophthalmic goiter and "toxic adenoma," there is a striking contrast in regard to the quantity of iodine required and the number of days necessary to accomplish the same result in the two groups.

Probably the most important factor in determining the relative iodine tolerance of exophthalmic goiter and "toxic adenoma" is the difference in degree of glandular hypertrophy and hyperplasia of the thyroid in the two groups.

In view of our experience, we feel justified in recommending the administration of iodine as a measure preliminary to operation in all cases of toxic goiter whether the thyroid be adenomatous or nonadenomatous.

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DISLOCATION OF THE PERONEAL TENDONS: RETENTION BY PLASTIC OPERATION ON THE EXTERNAL LATERAL LIGAMENT*

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While partial dislocation of the peroneal tendons evidenced by the presence of a prominent curved cord lying over the posterior edge of the lateral malleolus is common, complete dislocation is relatively infrequent. The former most often goes unnoticed because it produces no symptoms, but the latter is painful and disabling.

Compared to dislocation of other tendons passing joints, dislocation of the peroneals, if not the most frequent in occurrence, is certainly the most frequently recognized. Dislocation of the tibialis posticus upon the mesial malleolus is rare, but the tendon of the long head of the biceps not infrequently wanders out of its groove upon the adjacent surface of the lesser humeral tuberosity, probably much more often than its displacement is recognized during life. (A. W. Meyer.)

The mechanism of retention of the peroneal tendons in place, as they curve around the lateral malleolus as on a pulley, consists of three elements—the groove on the posterior aspect of the malleolus, the sling arrangement of the fibers of the external annular ligament, and the fact that the groove of the cuboid through which the long peroneal tendon passes is mesially located with reference to the lateral malleolus, so that tension on the tendon tends to keep it in place, while the short peroneal is held by the long tendon which crosses over it.

The groove on the malleolus is often so shallow as scarcely to deserve the name and thus can offer but little resistance to dislocation. The sling of the external annular ligament, the retinaculum peroneorum superius and inferius of Henle, is the principal

* Read by title.

means of retention, but its elements are delicate, even flimsy, so easily may they be ruptured. The curve of the long peroneal tendon, with concavity directed mesially, is entirely obliterated in exaggerated pronation of the foot, so that in this position there is practically nothing to hold the tendons in place except the annular ligament.

Thus dislocation of the peroneal tendons is generally associated with flat-foot or pronation and may occur gradually by stretching of the flimsy ligamentous slings, a wandering of the tendons out of their groove upon the malleolus. They take with them their sheath, which at this point is a single sac, being divided only below. In exaggerated cases the thecal membrane spreads out over the entire malleolus. Or in some cases, possibly the majority, dislocation results from a sudden strain, as in jumping, particularly if at the time the foot is in pronation, when the delicate attachment of the annular ligament to the malleolus is torn. Some swelling occurs, perhaps some discoloration, and the patient thinks he has sprained his ankle. The dislocation is apt not to be discovered until later. In the process of healing the thecal membrane grows out over the malleolus and the adjacent inner surface of the superficial fascia. The volume of the sheath thus becomes greatly increased, and redislocation of the tendons occurs as the result of the slightest trauma in the so-called habitual dislocation. The foot is comfortable as long as the tendons are in place and the patient may go about his business quite normally, when suddenly without warning and generally the result of taking a quick step when the foot is in pronation, the tendons slip out upon the surface of the malleolus, often with disabling pain. The trauma of walking may set up tenosynovitis, which is also painful. If the groove on the malleolus is particularly shallow, or the surface oblique in the favorable direction, dislocation of the tendons may take place in violent effort, as probably occurred in Case I, reported below.

Sometimes only the tendon of the long peroneal is dislocated, but generally both tendons go together. The tendon sheath, being exposed to unusual trauma, may become inflamed.

In the case of acute traumatic dislocation early reduction followed by retention by means of pad and bandage, and the foot held in supination in plaster-of-Paris, may be efficient in preventing redislocation if retention is maintained long enough for

cicatrization to reattach the annular ligament to the malleolus; but in dislocation by gradual wandering of the tendons, as in high degrees of pronation and in other forms of habitual dislocation, open operation is required.

Two procedures are recommended: The operation of Koenig and Kraske, which consists of lifting up a periosteal-bone flap from the surface of the lateral malleolus, taking care not to tear its thin posterior attachment, *i. e.*, on the edge of the groove, folding the flap backward over the tendons, attaching its free edge by suture to the ligamentous apparatus behind and beneath. Since the thecal membrane has grown over the malleolus, this periosteal-bone flap will be lined with smooth membrane on the side which is placed next the tendons. This operation is generally recommended and is said to give satisfactory results in most cases. Its weak point is that the attachment of the flap along the edge of the groove is very delicate and will not offer much resistance to dislocation. The other method is deepening of the groove in the malleolus. This requires elevation or removal of the thecal membrane from its surface. Practically this membrane is so thin that it cannot be elevated and the tendons then ride over uncovered bone.

In two cases the writer has used with gratifying permanent result an operative method which seems to have the advantage of being more positive, in that it is an attempt practically to rehabilitate the torn slings of the annular ligament, taking advantage of the naturally strong attachment of ligament to bone. The method is as follows:

The lateral malleolus is bound to the tarsus by the so-called external lateral ligament. This is described as consisting of three parts or fasciculi—sometimes described as three separate ligaments, an anterior and a posterior fasciculus, both attached to the talus, and a middle fasciculus attached to the calcaneus.

The peroneal tendons lie directly in contact with the superficial surfaces of the posterior and middle fasciculi. The latter extends directly backward and inward from the tip of the malleolus 4 or 5 cm. to its attachment on the lateral surface of the calcaneus. Its thicker portion is 4 to 6 mm. wide.

Through a suitable incision this middle fasciculus is readily exposed without injury of any important structure; with a little care the external saphenous nerve which accompanies the vein of

the same name may be avoided, but it is, after all, merely a sensory branch of little importance. The ligament is split into two halves; one of them, preferably the superior, is divided at its insertion on the calcaneus and dissected free as far as its insertion into the tip of the malleolus; its end is then passed between the peroneal tendons and the malleolus and is brought out superficially; its free end is then sutured with fine kangaroo tendon or silk to the uncut portion of the ligament, making a loop or sling which will hold the tendons securely in place.

If it were practicable to pass the detached half of the fasciculus in front of the sheath of the tendons, the arrangement would be ideal, but the wall of the sheath is too delicate and too firmly attached to the bone to be dissected off without tearing it. As a matter of fact, however, there seems to be no disadvantage in the lack of thecal covering of the loop, for the wall of the sheath grows over any adjacent connective-tissue surface, especially under conditions of pressure and sliding of the tendons—witness its spread over the surface of the malleolus in old dislocation—and may be relied upon speedily to heal any wounds which may have been made in it and effectively to line the loop. At least in the writer's two cases there was no disability or discomfort which could be attributed to adhesions of the tendons.

Case I.—W. F. L., a lineman, aged twenty-five years, in September, 1917, fell from a telegraph pole, turning his ankle, as he said, so that the sole pointed inward. For supposed fracture of the tip of the lateral malleolus the foot was put up in plaster-of-Paris for three weeks. A week after removal of the cast it was noticed that the peroneal tendons slipped over the outer surface of the malleolus. Some relief was had from strapping the ankle with adhesive plaster, but, notwithstanding, dislocation continually recurred and patient was obliged many times a day to stop work, remove his shoe, put the tendons back into place, adjust a pad and restrap.

In November, when first seen by us, there was no radiographic evidence of fracture of the malleolus, the tendons slipped forward nearly to the anterior edge of the malleolus on the slightest movement of active pronation.

Operation disclosed the fact that the thecal membrane covered the entire lateral surface of the malleolus. The redundant portions of the sheath were cut away and a sling made of the superior half of the middle fasciculus of the external lateral ligament, as described above. The

sheath was repaired as far as possible, but very imperfectly, and the annular ligament sutured. Plaster-of-Paris in supination nine days, with subsequent elevation of the inner side of sole and heel about $\frac{1}{2}$ cm. Twelve weeks later there had been no recurrence of dislocation and no disability.

Case II.—M. W., a young woman, aged twenty-nine years, tall, active, athletic, in March, 1923, mounting a horse, felt something snap in the left ankle. The peroneal tendons had slipped over the lateral surface of the malleolus. They were replaced and the ankle and foot strapped at once. Patient was allowed to walk, but with ankle held stiff for five weeks. Dislocation recurred repeatedly as soon as support was omitted.

Operation as above. Sheath here also found to cover the entire lateral surface of the malleolus. It contained a small amount of yellowish fluid, the color doubtless due to old blood pigment, thus proving the trauma. Ligament split and sutured as above with fine kangaroo tendon; in plaster-of-Paris eight days; slight tilting of sole of shoe thereafter. Five weeks later there had been no recurrence of dislocation, patient walking without limp, going up and down stairs without discomfort. Three years later, no recurrence, no disability.

SEPTIC OSTEOMYELITIS OF THE BONES OF THE SKULL AND FACE

A Plea for Conservative Treatment*

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In this paper it is proposed to consider only inflammation or death of bone due to infection with a pus-producing organism, but three somewhat distinct clinical entities will be included:

1. The condition commonly called "ulcerated tooth."
2. Frank ostitis and necrosis of the dentigerous bones.
3. The spreading ostitis and necrosis that may accompany or follow pus infection of the paranasal or paraäural sinuses.

Our reasons for including these in one group are:

1. They are each essentially a bone infection, and though showing certain individualizing characteristics, the pathology is basically the same, the reaction differing rather in degree than kind, and we believe the treatment of each is essentially the same.
2. Further, while the lines that demarcate the typical cases in each group can be sharply drawn, there occur apparently borderline cases that are not so easily catalogued, and the close anatomical relation of the parts affected facilitates this grouping.

* Of the matter herein contained, the "ulcerated tooth" was made the subject of an editorial presented previously in *Surgery, Gynecology and Obstetrics*. The case reports referring to osteomyelitis of the jaws were reported recently in *Surgical Clinics of North America*. The section relating to osteomyelitis of paranasal-sinus origin is based upon part of the data that is being gathered for a more formal presentation that is proposed to be made at a future date before the American Laryngological Association.

General Conservatism in Treatment of Pus-infected Bones. There is an Italian proverb which says: "He who goes slowly goes safely; he who goes safely goes surely."

The above can be taken as a safe guiding text in the treatment of simple osteomyelitis of at least the jawbones. The general plan of good surgical treatment of simple purulent osteomyelitis was firmly established many years ago, and, except as influenced by the presence of the teeth, its treatment when affecting the skull and jaws differs little from that of any other bone. Due partly to the superior resistance of the face and mouth tissues to most infections, and partly to the readiness with which spontaneous drainage is established around the teeth, in the jaw the disease has not the inherent fatal tendency that may characterize it elsewhere. On the other hand, the deformities that result, most commonly from ill-advised surgery, can be little short of ghastly. In certain spreading infections of calvarium this is not true. Resulting deformities are not marked, but disease is frequently fatal.

The accepted treatment of osteomyelitis in general, as handed down to the senior collaborator some thirty odd years ago, consisted in, first, the early establishment of drainage of the focus with the least possible operative trauma; second, waiting until the virulence of the infection had subsided, the dead bone had spontaneously separated and sufficient new bone had been formed to maintain continuity before attempting any radical operation; third, at the proper time to remove all fragments of dead bone with limited damage to granulations lining their beds, and, where practicable, to remove all edges of live bone that overhang bed so that soft tissue can drop into and fill these defects. The latter can often be facilitated by fashioning appropriate and well-nourished flaps of skin and subcutaneous tissue; muscle may be included.

When the above program is faithfully and intelligently carried out the disease will seldom prove fatal or progressive, and successful sequestrectomy followed by permanent healing will usually be accomplished by one operation. When dealing with cavities in cancellous bone near joints the propriety of the latter part of the procedure may be questioned by some surgeons who are accustomed to filling such defects with Mosetig's bonewax, gauze or some other substance, but there are comparatively few instances

where a live flap cannot be advantageously substituted for these foreign fillers.

Many advantageous refinements of technic have been more recently worked out. Especially desirable are those that strive to raise the resistance of the tissues, or to protect the granulation-lined cavities from secondary infection, or to sterilize them by noncorrosive lotions; but any radical departure from the basic principles just cited is apt to be followed by disaster or embarrassment in one form or another. This older plan of conservative treatment has in late years been subjected to very forceful attacks, first by the operating dentists and exodontists, later by the rhinologists. The following will be an attempted recapitulation of the facts as we have observed them:

Portals of Entry. If we are to consider prophylaxis against spreading septic infections within the jawbones, we must go into the etiology at least as far as portals of entry.

In the upper jaw the infection may apparently be by way of the nasal mucosa or antrum. (See Case Reports I and II.) In either jaw it can unquestionably be blood carried from a distance, as is common in osteomyelitis in other bones, but where infected teeth are present it may be difficult to draw absolute conclusions. Commonly, in children, the necrosis apparently follows the extraction of a tooth, but in at least some of these the bone infection may have caused the symptoms for which the tooth was drawn. When the symptoms are first noted immediately after the treatment of a quiescent nonvital tooth, this rather suggests a preexisting periapical infection which may have been present for years. When such an infection involves a tooth with intact walls, it must be considered as being of blood-borne origin. In one child the necrosis followed an extraction during the acute stage of a fulminating infection, and we have had such a history in a few adult cases; but it has been our observation that trouble following extractions under such conditions is more apt to result in diffuse infection of the soft parts and occasionally the death of the patient rather than an extensive necrosis of the bone.

While it is not safe to reach a *post hoc ergo propter hoc* conclusion in every or even the majority of cases, it is, nevertheless, all things considered, a safe clinical bet that prevention of decay and of injury to the teeth will prevent possibly quite a large percentage of these bone infections.

There may occur a low-grade bone infection that forms little pus, more or less pain and a variable amount of swelling. It is apt to attack different parts of the same jaw, or both jaws simultaneously or consecutively, throwing off a sliver of bone here or loosening a tooth there, going on for a long time without coming to any definite conclusion or without showing any very definite head for surgical attack. The patient meanwhile may show various signs of intoxication.

In the earlier literature the belief that this type of necrosis was dependent on syphilis is quite common, but such an etiological factor now appears to be rather rare. The former belief was, in the past, quite firmly held by the senior writer of this paper. We find but few of the Wassermann reports of these cases to be positive, and it may be that the apparently good results from antisyphilitic therapy were due either to some antiseptic quality in the medication or to the delay entailed which gave further time for the development of specific antibodies.

In the bones of the calvarium the paranasal sinuses are most commonly the portals of entry.

The "Ulcerated Tooth." The above term commonly designates an acute exacerbation of a previously quiescent perapical infection; this naturally and by common consent will call for the ministration of the dental surgeon. The attack may terminate or be terminated in one of several ways: After two days of suffering an abscess may perforate the bone, causing the typical "gum boil," or, less commonly, it may burrow out alongside of the root of the tooth. The dentist may get drainage through a root canal or an attempt may be made to abort the process by extracting the tooth. The latter treatment has the virtue of precluding future attacks and may be followed by quick recovery, by a more or less protracted or stormy convalescence or occasionally by death from general sepsis.

It was an old teaching in dentistry that these teeth should not be pulled during the period of acute swelling, and there is usually sound clinical observation back of these older teachings. With more modern and antiseptic technic the general trend is to substitute active intervention for cultured conservatism, and, above all, with the advent of the "exodontic" specialist this older teaching lost precedence. The attempt to establish free drainage and at the same time remove the supposed focus is apt to appeal more

strongly to the new dentist. This procedure has been compared to the removal of an acutely diseased appendix; but the simile is inapt, because the appendix is the focus of the infection, while the tooth is at this time but an inert plug. In their results the two procedures do not parallel, and the observer standing on the side line who takes occasion to sort over the wreckage is apt to conclude that the average results do not justify the extra risk inherent to early extraction. In a certain small percentage of cases the reaction is accompanied by an increase of symptoms, or is followed by abscess formation or extensive bone necrosis. In addition to this, some young or apparently robust people will die from general sepsis following extraction of an "ulcerated tooth" in the acute stage. On the other hand, death following the conservative plan is extremely rare, and, except among young children, cervical abscess or extensive bone necrosis is uncommon where the tooth and the bone are spared the trauma of instrumentation in the acute stage of the infection.

This may seem difficult to explain, but a study of the history of a typical case will at least furnish food for thought.

The "ulcerated tooth" is a culmination of an infection that has been present for an indefinite time, possibly years, without giving more than mild or unidentified symptoms. Often the root canals of the damaged tooth had long ago been sealed by the dentist, and it is usually difficult to assign any logical cause for the explosion. About the simplest explanation is the assumption that a disturbance of the balance between virulence and resistance has occurred which permits the hitherto imprisoned bacteria to successfully attack the confining barriers. Such a period of low resistance may be the reason why the trauma of an extraction may not be well tolerated at this particular time. This type of acute osteomyelitis should hold more than an academic interest for both the physician and the surgeon, either of whom must occasionally help a patient to choose between the man who offers immediate and permanent relief from a jumping toothache by a painless extraction under gas, and the old-fashioned dentist who prescribes quinine, phenacetin, the mustard foot-bath, the fig poultice, and who may attempt to establish drainage by the somewhat painful process of opening a root canal. The former may be the more brilliant procedure, but we can still give the conservative man our moral support with the assurance that his plan is the safer. Besides,

the physician can always add sufficient morphine at least to ease the time of travail, and possibly he may shorten it by an incision and a stripping-up of the periosteum at the likely site of perforation

At a later period when the balance between virulence and resistance has been reëstablished in the patient's favor, extraction of the tooth is not only safe, but is better surgery than the most effective dental restoration.

This taboo against immediate extraction applies only to the period of acute local symptoms, evidenced chiefly by swelling of the neighboring soft tissues and by the pain and tenderness that are characteristic of early osteomyelitis, not to the subacute stage in which discomfort, low fever, adenitis, malaise, rheumatism or joint infections, etc., may evidence chronic infection. If, however, an extraction under these latter conditions is followed by a severe local flare-up, then there may be good reason to go slow on repeating the insult.

Necrosis of the Jawbones. Simple osteomyelitis of the upper jaw resembles the same affection in other cancellous bones, in that it is of more rare occurrence and the necrotic areas are apt to be less extensive.

In either jaw the need for early artificial drainage of the focus is seldom urgent, though confined or pocketing collections of pus should be liberated by internal or external incision when detected. It is maintained by many of the dental profession that the application of external heat in the early stages fosters the formation of an external fistula. We have done the thing that has given the patient the greatest comfort, whether it has been the use of heat or cold.

The very objectionable odor that is given off from some older cases of jaw necrosis we have found to be controlled by one or two thorough irrigations of all fistulæ with a quantity of 1 per cent solution of formalin (2.5 per cent solution of the 40 per cent preparation of formalin) in water. The stinging pain of the injection does not last long, but can be modified by a previous irrigation with 1 per cent novocain.

It is difficult to set an exact time, but in the ordinary straightforward osteomyelitis of the jaw the dead bone will usually have separated itself in ninety days, and by this time a strong involucrum will have formed that will permit the removal of the sequestrum without changing the normal contour of the jaw. It has

been our observation that instrumental manipulation of the bone, of at least the lower jaw, before the infection has lost its virulence, is very apt to be followed by further extension of the necrosis. In certain cases in which the periosteum has been stripped off or other attempts have been made to dislodge a piece of dead bone before it has become loose, we have found different segments in the same jaw that have been dead for different periods of time, so that one piece may be loose, "worm-eaten" and encased in a strong involucrum, while a neighboring piece may have but recently died and not yet detached, or if detached, be surrounded by so little new-bone formation that after its removal the jaw may collapse at this segment or the bone may fail to regenerate. (See Case III.)

In some cases of extensive necrosis it may be very good surgery to divide the radical operation into several steps, thoroughly cleaning out one area at a time. We have found that the surest way of cutting the necessary number of radical operations to the minimum is to give a full three months between the original infection, or any subsequent instrumentation, before attempting to remove the dead bone, and then, if it is not found to be "worm-eaten" and surrounded by a definite bed of granulations, to wait another three months before making a subsequent attempt.

For other reasons it may be desirable to wait very much longer than three months before disturbing dead bone that is in close relation to developing teeth. Up to a very few years ago it was our practice to remove not only all loose "worm-eaten" fragments of bone, but all teeth and tooth-buds that were definitely within the necrosed area, on the theory that they must be dead and could only act as harbingers of infection. It was Dr. Virgil Loeb, of this city, who first called our attention to a case of which he had a very striking series of x-rays. The first showed certain partially developed teeth to be very definitely surrounded by dead bone. Dr. Loeb had not done a radical operation, but had waited for the dead fragments to be thrown off spontaneously, with the result that the tooth-buds continued to develop, and the slightly deformed teeth had become fixed in the newly-developed jawbone, as demonstrated by a radiograph. Since then we have verified this result on several of our own patients, and it is now our custom in treating necrosis of the body of the jaw in a child to furnish

drainage as indicated, watch closely for kidney damage, and, if the general condition permits, to do no radical operation on the alveolar portion, but wait for the fragments to be thrown off spontaneously.* Fortunately, in children in the tooth-bearing areas the dead bone is not apt to become deeply sequestrated, as may happen in the ramus, but this does not hold true with adults. (See Case IV.) The preservation of the tooth-buds is of tremendous advantage. In areas where they are completely lost the new bone is apt to be short to the extent of very serious deformity. (See Cases V and VI.) If one first or second molar is preserved, it may save excessive retraction of the regenerated jaw, but preservation of the third molar bud will not do this. (See Cases V and VI and VII and VIII.)

Much more disastrous than the removal of tooth-buds is the complete lack of regeneration that may follow the too early removal of the dead bone or the attempt to control infection by resecting the original live bone. (See Cases VI and IX.) We know of one case in which the entire lower jaw is lacking from repeated attempts of this kind, and there never has come under our notice a single instance of failure of regeneration of the mandible following a simple osteomyelitis that had not been subjected to early energetic bone surgery. (See Case IX.)

Plan of Removal of Sequestra. In the lower jaw the incision is made along the alveolar process; or (as was necessary in Case IV), an incision can be made on the skin surface along the lower border of the mandible and part way up the posterior border of the ramus from one side to the other. For necrosis of the ramus in adults, for locating dislodged spicules in the sigmoid notch, the posterior part of the incision just described is most appropriate.

After the sequestrum has been exposed, slip a curet under the various fragments and ease them out, the cutting edge turned toward the dead bone and not toward the involucrum. This plan is usually sufficient, even for a totally necrosed ramus and condyle. When the sequestrum is buried in hard bone, it may be necessary

* Cryer quotes Percy, of Paris, in 1791, as having observed that teeth in the necrosed area of bone finally recovered their original solidity and were firmly implanted in the thickened gum. Necrosis following extraction was known as far back as Heister's time (1710), and he warned against extractions in inflammatory affections of the jaws.

to chisel away one wall of the involucrum before the above can be carried out. Where the pockets are multiple they must be dealt with individually, but all may be cleared out at one operation. A curet makes a most efficient persuader, and it should be used as a tractor, an elevator or as a tool to carve away overhanging involucrum, but as a bone-scraper only when hunting for hidden fistulæ that might lead to other sequestra. Bone-scraping as a habit is a pernicious practice, and is to be deplored even when necessary. The search for small, deeply-buried spicules may be greatly facilitated by a preliminary injection of the fistula with methylene-blue solution.

In every case every piece of dead bone must be removed or so treated that it can be discharged spontaneously, otherwise the wound will not heal permanently. It is for the lack of such treatment that many cases remain unhealed for a period of years that otherwise might have been cured in a few months.

In the upper jaw the sequestra are apt to become buried in scar. In seeking to remove these, it is a better plan to dissect out the scar than to try to find the individual pieces of bone. This is sometimes true in regard to fragments in the neck that have worked down from the lower jaw. (See Case X.)

In all the preceding the emphasis has been on time-consuming conservatism, but when conditions become right for radical operation, one should act boldly and efficiently. The necrosed alveolar process will be thrown off spontaneously or with very little help, but pieces of dead bone in the body or ramus will become encased by the living bone unless surgically removed and can remain indefinitely as a focus of infection. (See Cases XIII, XIV and XV.) If, after the sequestrum has been removed, the pocket in the involucrum is very deep, surrounded possibly by overhanging walls, such a cavity may take years to scar over, and, therefore, should not be left in this condition.

Conservatism the Most General Choice. The literature still shows the old division between those who advocate early conservatism *versus* those who promise to abort the disease by the earliest possible radical surgery. The position of the latter, who are still in the minority, is supported neither by new arguments nor any adequate number of convincing case reports.

Case Reports*

Cases considered in this paper do not include those primarily due to open injury to the bone; this rules out all open fractures.

The case reports are taken from cases treated since 1916 at Barnes Hospital, St. Louis Children's Hospital and St. Louis Mullanphy Hospital, because since that time the plan of recording histories has been more uniform.

Case I.—Girl, aged two and a half years. Infection of maxilla, occurring most likely through antrum during course of diphtheria, treated expectantly, followed by recovery.

During course of diphtheria swelling of right side of face occurred with, later, a discharge of pus. Removal of loose pieces of bone from outer surface of maxilla five, six and seventh months later, with entire recovery.

Case II.—Baby, aged six and a half months. Infection of maxilla and orbit probably of antral origin. Early bony extension, bony incision for drainage—death from pneumonia.

Well-nourished baby developed severe general symptoms of an infection and a swelling of the upper gum and hard palate, which later extended to face and orbit. Seven days later, in an effort (probably ill-advised) to obtain drainage, entrance was made through the palate into orbit and antrum, removing several infected tooth-buds. Nine days later the orbit was again entered and more pus obtained, but the child died of pneumonia. This is the only case in the whole series of jaw cases that terminated fatally, and we feel that the child's chances of recovery were not helped by the early bone-cutting operation.

Marx, in 1920, called attention to the ocular lesions consecutive to osteomyelitis of the upper jaw in infants. Nord, in 1924, again called attention to it, and stated that the correct diagnosis was often missed. He gave as treatment a conservative outlet for pus and waiting for spontaneous separation of the sequestrum, with which plan we are in full accord.

Case III.—Boy, aged five years. Necrosis of the mandible, which probably antedated tooth extraction; bone-cutting operation followed by a further spread of the necrosis; too early removal of necrosed ramus, followed by failure of regeneration of the ramus; persistent fistula due to retained dead, partially developed crown.

* The "follow-up" of these cases has been made possible only by the coöperation and work of the Social Service Department.

A *B*

FIG 1.—Case III.

FIG. 2.—Case III.

The boy had two molar teeth on left side extracted on account of swelling of the face, and piece of loose bone was removed from the gum in the clinic three days after the extraction. This suggests that the necrosis had antedated the tooth extraction by some considerable time. Three and a half months later an operation was done that seems to have included raising some periosteum and curetting the involucrum. Eighty days after this last operation, the face had again swelled and a very recently necrosed ramus was removed, showing little destruction at the time of operation. This early removal of the ramus was not followed by regeneration. Persistence of the fistulæ necessitated two further operations; at one a dead tooth-bud was removed from the ramus, and at the other a dead, partially developed crown.

The whole course, as viewed in retrospect, suggests a case of low-grade bone infection with slow separation of the dead fragments, and that the infection was spread and new areas of necrosis occurred as the result of the attack on the live bone. The premature removal of the ramus fragment was, in this case, only an incident; but if a failure of regeneration had occurred in the body similar to that in the ramus, it would have been a catastrophe requiring a subsequent bone-graft. There is some retraction of the jaw as a whole on the affected side, as shown in the photographs (Fig. 1, *A* and *B*).

Fig. 2 is of an *x*-ray taken June 16, 1925, eight years and seven months after the last operation, and at the age of thirteen years. The unerupted lower third molar is seen to be in proper relation to its opponent above and to the remaining part of the anterior border of the ramus. This would suggest that at least part of the retraction is due to shortening in the regenerated part of the body. It plainly shows the lack of the ramus, and also shows a somewhat deformed third molar bud.

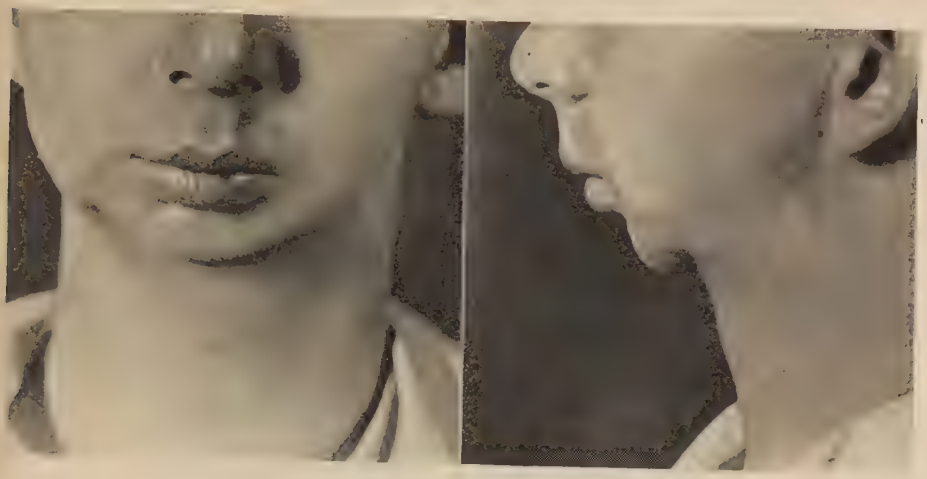
Case IV.—Man, aged thirty years. Extensive necrosis with multiple sequestra. First seen three months after original trouble, and found to have widespread necrosis, with between twenty and thirty pieces of sequestrum buried in separate pockets in the involucrum in the body and rami (Fig. 3).

The operations were done over a period of time extending from seven and a half months to two and a half years after the onset of symptoms. There was recovery without diminution of the size of the mandibular arch, but all the teeth were lost. This case would tend to show that the long-delayed operation in adults would not save the teeth. However, the jaw is already developed and the saving of the teeth is not as important as it is where their presence is to be counted on to help preserve the length of the developing jaw.

Fig. 3.—Case IV.

Fig. 4.—Case V.

CASE V.—Girl, aged twenty years, who had loss of all tooth-buds at five years as a result of necrosis. The measurements made at the time of examination showed the body of the lower jaw to correspond to a normal jaw of about five years. We have observed other similar cases. A third molar is present, but this has had no effect on the development of the jaw (Fig. 4).



A *B*

FIG. 5.—Case VI.

A *B*

FIG. 6.—Case VI

CASE VI.—Man, aged twenty-two years. Failure of regeneration of jaw after curetting operation on lower jaw for necrosis one month after a tooth infection. Operation for necrosis at eight years of age. First seen at twenty-two years. The third molars were the only teeth that had developed, with the resulting lack of development of mandible. (Fig. 5, *A* and *B*, showing deformity.) Fig. 6, *A* and *B*, after cartilage and skin grafts to permit use of tooth-carrying prosthesis. Prosthesis made by Dr. James A. Brown.

FIG. 7*A*.—Case VII.

CASE VII.—Girl, aged ten years. Extensive necrosis of mandible following filling of a tooth. Sequestrectomy delayed eight months, with preservation of some molar teeth that were in the necrosed area and preservation of almost the normal length of the jawbone.

Two weeks after the filling of a tooth the tissues over mandible swelled, first on right, then both sides; pain, abscess opened, and loose anterior teeth extracted two months later. Pus continued to drain and was first seen at the Washington University Dispensary two months after first symptoms. *X*-ray condition at this time is shown in Fig. 7 *A*.

It shows complete necrosis of the left side of body and ramus, the necrotic bone breaking into fragments, a fully erupted lower first premolar and a partially erupted second premolar and second molar. New-bone formation of the developing involucrum can be seen on the mental part of the body, but an attempt to remove the sequestra would have resulted in the loss of all associated teeth and most likely in a failure of development of new bone. Drainage had continued for

Fig. 7*B*.—Case VII.

five months, when the child was sent to the St. Louis Children's Hospital to determine if the continuous suppuration had or was likely to cause kidney or other vital damage. Dr. Marriott advised that he thought it safe to continue without at the present time removing the sequestra.

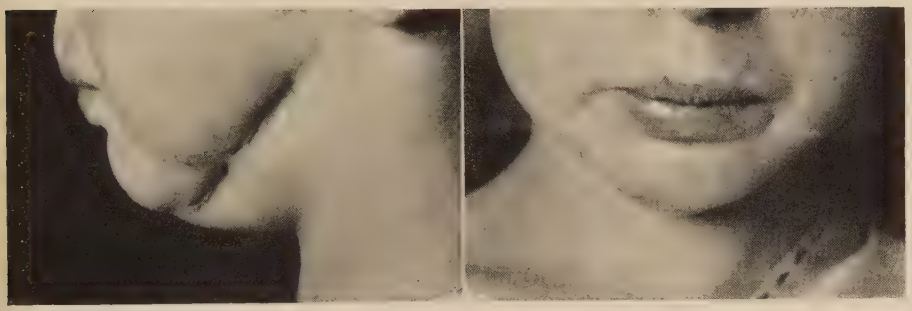
Fig. 7 *B*, shows a well-developed involucrum, and that the second premolar has either erupted further or is being thrown off. The devel-

oping crown of the third molar is also apparent. The masses of the sequestra are more clearly defined. It was in hopes of preserving some of the developing teeth that the sequestra were not removed at this time. The child was kept under careful observation, with repeated blood and urine examinations, three months longer. Eight months after first appearance of the symptoms. Dr. Earl Padgett (then an associate in this service) removed the sequestra and rongeured away

Fig. 7*C*.—Case VII.

overhanging bone. Child made a good recovery and the condition one and a half years later is shown in Fig. 7 *C*. Both premolar teeth have been lost, but the second and third molars have continued to develop about normally. There is a good strong body, but the only evidence of an alveolar process is about the second molar, which will be seen to be in approximately normal relation with its fellow above. The base of the third molar is situated a little farther back than normal,

but this seems to be the rule in jaws that have been necrotic. On the report received as to present condition there was a question as to the possibility of a suppuration about one tooth, but there is no evidence of it in this radiograph. The important point is that the jaw seems to have continued to grow, and, though there has been some question about its length, it is no shorter relatively than not infrequently occurs in children of her age who have had no bone infection, and with the normal molar occlusion preserved there is little likelihood of any increasing deformity developing. Conditions on the right side about matched those of the left, but here the second molar was finally lost and the first premolar was preserved.



A *B*

FIG. 8.—Case VII.

Fig. 8, *A* and *B*. (Photographs taken July 6, 1925.) The scar can be taken care of by removing the involved area down to the bone, undermining the edges and then suturing them together.

CASE VIII.—Boy, aged twelve years. Tightening of teeth that were loose in an infected jaw. *X*-ray three months after onset showed necrosis of right body back to second molar, with the first molar rising out of its bed, the cuspid, two premolars and second molar in the necrotic area. The final sequestrectomy in this case was done four and a half months later, or eight months after the beginning of the trouble. Six months later a report was received from the dentist that all the teeth mentioned were in place and tight except the first molar, and also that a third molar had erupted.

This case is similar to Case VII in demonstrating the possibility of saving teeth and jaw symmetry in children by waiting long enough to remove the sequestrum. The reason for this is that the tooth-bearing area in children does not sequestrate.

CASE IX.—Woman, aged thirty-three years. Lack of development following extraction of teeth and some unidentified bone operations. Operations done at age of twelve years following necrosis from extraction of a tooth.

A *B*

FIG. 9.—Case IX.

When first seen at age of thirty-three years she had a little nubbin of bone in the neighborhood of the right glenoid fossa, and all of the lower jaw on the left as far forward as the region of the cuspid tooth. There was also some failure of development of the superficial tissues corresponding to the missing bone, and she had several bad teeth, which were pulled.

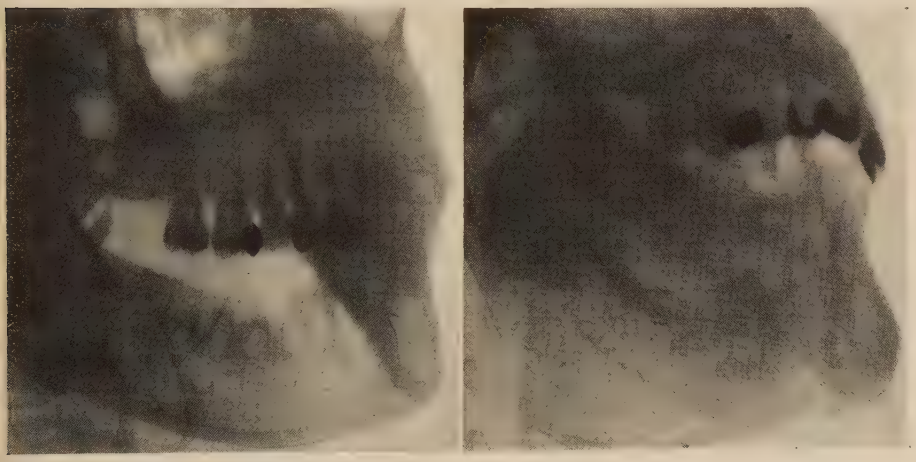
A *B*

FIG. 10.—Case IX.

FIG. 9, *A* and *B*. Showing deformity of jaw and lack of soft parts.
Fig. 10, *A* and *B*. Appearance after skin and rib grafting and with prosthesis in place. Prosthesis made by Dr. James A. Brown.

CASE X.—Boy, aged eight years. Maxillary necrosis related to tooth infection; repeated early operations failed to control symptoms.

Necrosis of maxilla following tooth extraction. During the next few months there were five operations on the bone in an attempt to control the suppuration. Eight months after the first appearance of symptoms there was a discharging fistula under the outer part of the left lower eyelid in a scar attached to the infraorbital border. This scar mass was dissected out without identifying the sequestra and drainage established into upper fornix of the vestibule of the mouth. No bone fragments were found; skin incision closed with small drain in place. Child recovered with one mild return of symptoms about three months later. Report by letter eleven months later states child is well.



A *B*

FIG. 11.—Case XI.

CASE XI.—Woman, aged forty-eight years. Antisyphilitic therapy and sequestrectomy after two years.

Gland enlargement under jaw; tooth pulled, followed by persistent purulent discharge. Wassermann negative; only suggestion of syphilis the ostitis and two miscarriages without further pregnancies. Blood-pressure over 200. Antisyphilitic treatment was given. Two years later partial sequestrectomy done. Six months after this there was still *X*-ray evidence of dead bone, but there had been so much improvement that waiting longer was advised. Patient died that same month—apoplexy.

FIG. 11, *A* and *B*. *X*-rays taken seven months apart show a progressive bone involvement.

Case XII.—Man, aged forty-eight years. Wassermann negative, but some relief of symptoms of pain and infection by salvarsan therapy. Sequestrectomy finally done.

1919, soreness and tenderness in all molars and they loosened. 1921, Molars pulled, three at a time, and later that year fourteen more teeth pulled at one time. Pain and discomfort continued, and in February, 1922, it became very severe, and thick yellow pus drained into the mouth. March, 1922, *x*-ray showed "moth-eaten" areas in all molar regions, most marked on right. The remaining teeth were loose and rough bone could be felt in the lower jaw. Pain was a severe symptom throughout the course of the disease. Diagnosis of necrosis was made with question of syphilis. Several Wassermann tests were negative, but in November, 1922, there was reported marked improvement after antisyphilitic treatment of several months. A clean easy sequestrectomy was done several months later.

Case XIII.—Colored man, aged twenty-five years. In 1909, at age of nine years, tooth broken in extraction, followed by pain and swelling. There was one operation one year later. He was treated for syphilis in 1924. First seen June 6, 1925. Left jaw swollen, poor occlusion, two sinuses present under jaw, and opening limited. Incision made in front of ear, all tissues turned down, including parotid. Dug out several pieces of dead bone, an impacted molar, and then the condyle and coronoid to give movement. The mouth was still bound with a scar, which was cut through.

Case XIV.—Woman, aged thirty-two years. Multiple extensive involvement of lower jaw with general symptoms that were not relieved until the last piece of dead bone was removed.

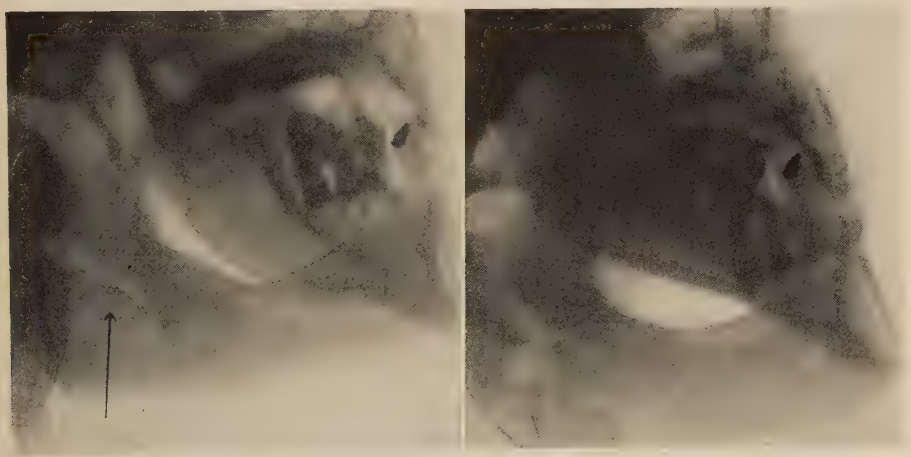
She was in perfect health until dental trouble arose after some arsenic had been left in a tooth for two weeks. There was local abscess and many teeth became loose. Widespread necrosis (Fig. 12 *A*) followed, many teeth were extracted, external drainage was done, and several sequestrectomies. There was local neuralgia, for which two nerve injections were done. There was also rheumatism, which cleared up, and there was a good general recovery after the last piece of dead bone was removed (Fig. 12 *B*).

Case XV.—Woman, aged thirty-four years. Following an inlay filling of right lower first molar, pain continued, and tooth was removed with excess trauma one month later. Pain and swelling for months, many small pieces of bone were removed, and jaws locked for months after removal of teeth on that side. Symptoms continued over a year

and a half. *X*-ray shown in Fig. 13 *A*, taken nineteen months after origin of symptoms, shows a spanner-shaped piece of dead bone lying

A *B*

Fig. 12.—Case XIV.



A *B*

Fig. 13.—Case XV.

in the substance of the body and lower part of ramus of the right mandible. The live bone embraces the sequestrum very closely. This, considered with the length of time the symptoms had been present,

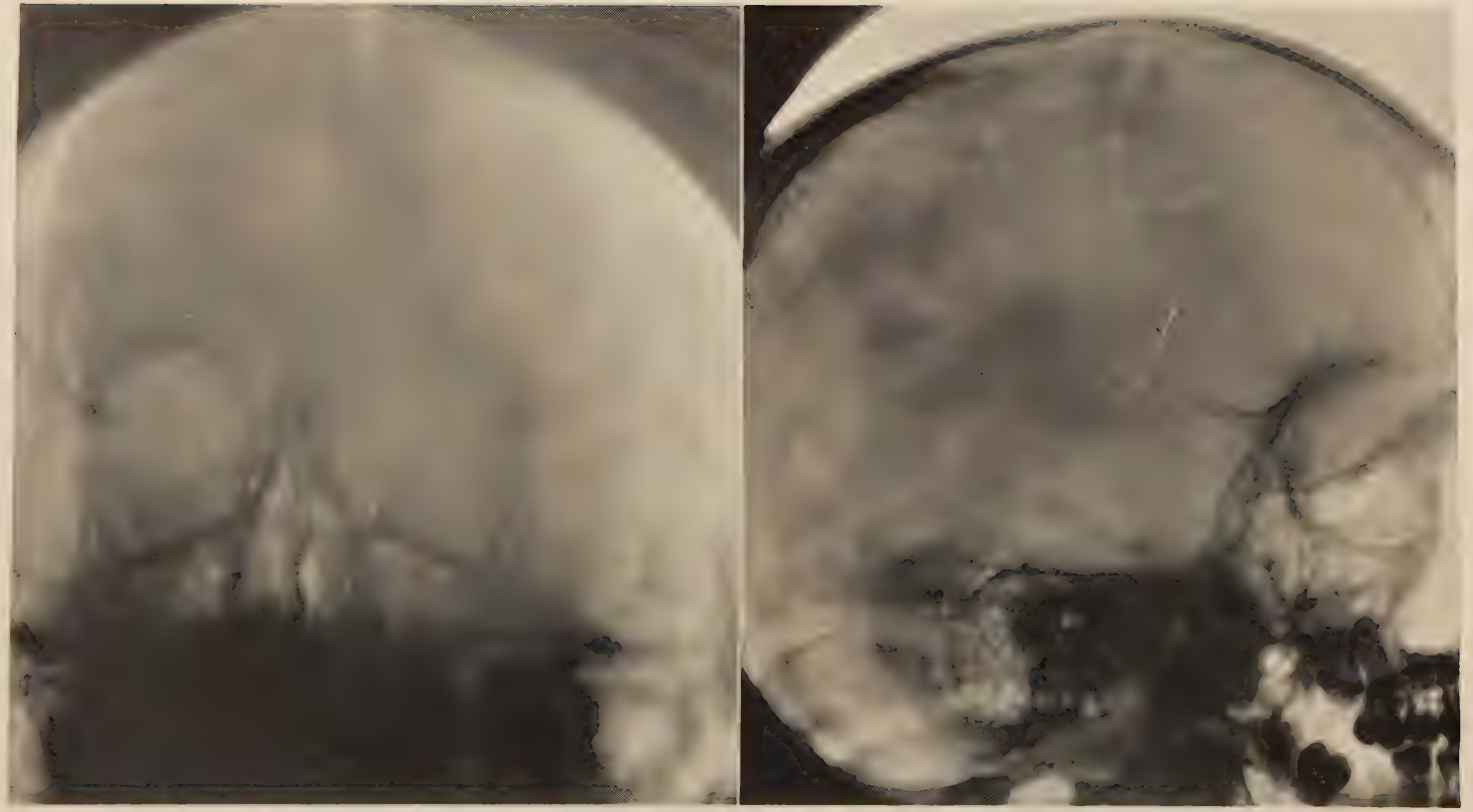


FIG. 14.—Case I*A*. Shows moderate-sized area of necrosis extending nearly to the vertex.

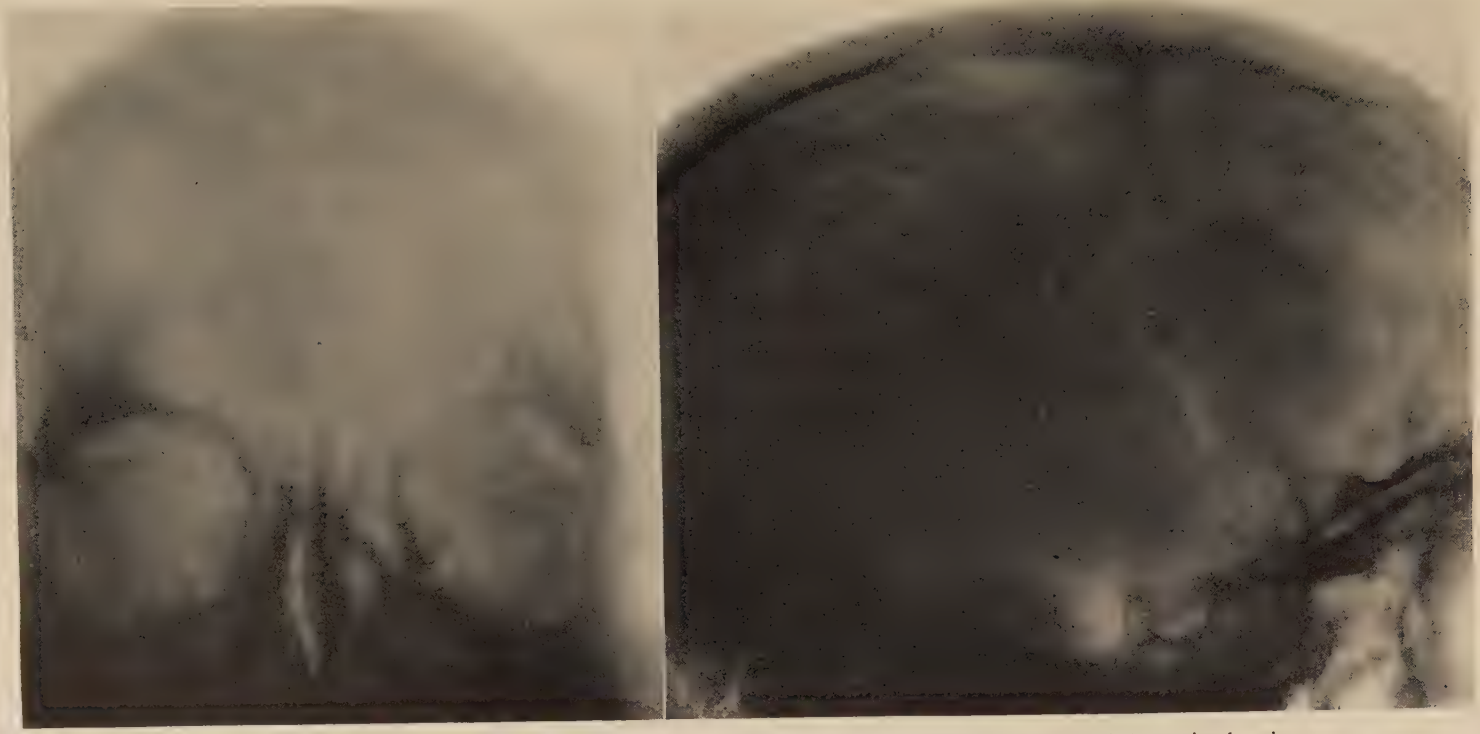


Fig. 15.—Case II*A*. Shows extensive rarefaction of frontal bone and some roughening of the parietal region.

and the lack of the mention of pus in connection with any of the various manifestations, rather suggests an infection with an organism that is not prone to cause suppuration. Most likely a streptococcus of low virulence.

Through an incision along the lower part of the border of the ramus and lower border of body the tissues superficial to the bone were raised and a subcortical sequestrum was laid bare and removed with a chisel. All granulation containing tunnels was converted into shallow grooves, and the soft tissues were sutured back into place with rubber dam drainage.

Patient discharged from hospital four days later, and at no time from then until the second operation was she entirely free from pain, swelling, slight fever, or discharge, though the wound had been reopened, packed, and wet dressings maintained. An *x*-ray taken three and a half months after the first operation (Fig. 13 *B*) shows the channel from which the sequestrum had previously been removed, but no further evidence of necrosis or sequestra. Four months after the first operation the whole area was laid bare, revealing the channels filled with granulations, wound packed, and Dakin treatment maintained for some time. The packing was continued for two and a half months, and when the wound had healed the pain and low fever continued. Later, the inferior dental nerve was injected with alcohol which for the time, at least, relieved the pain. No further observation of the case.

Diffuse Osteomyelitis of the Skull of Sinus Origin. Most of the recent rhinological literature supports very early and radical operative treatment of the necroses of the skull bones that may follow or accompany paranasal sinus infections, and a powerful array of facts and figures are cited to support this contention. This reversal of the more generally accepted rules for the treatment of osteomyelitis is not supported by our own clinical limited observation of these cases. Therefore, we have dug rather deeply into the literature of the subject reviewing over a hundred articles or detached case reports.

In reference to the literature these seem to be the facts as we have them. The first cases definitely reported as being of sinus origin were in 1899, one by Luc[1] and one by Tilley;[2] but there are a few reports of osteomyelitis of the skull giving practically the same clinical picture in the literature farther back.[3–8]

The radical writings consist of some twelve to fifteen articles, based partly on personal observations and largely on citations from an exhaustive article, in three parts, by Dan McKenzie, of London,

and at the time of the appearance of his articles, 1913, editor of the *Journal of Laryngology, Rhinology and Otology*. The latter writer deduced his conclusions from the study of 40 cases that he found in the literature, and from 1 case of his own. His findings were in the main that the cases (20 in all) in which the necrosis developed after a sinus operation, died regardless of treatment; that of the 21 cases developing spontaneously from sinus infection, 7 survived, and all of these 7 had been subjected to "appropriate surgical treatment," which, from his article, we are led to believe was early radical bone removal.

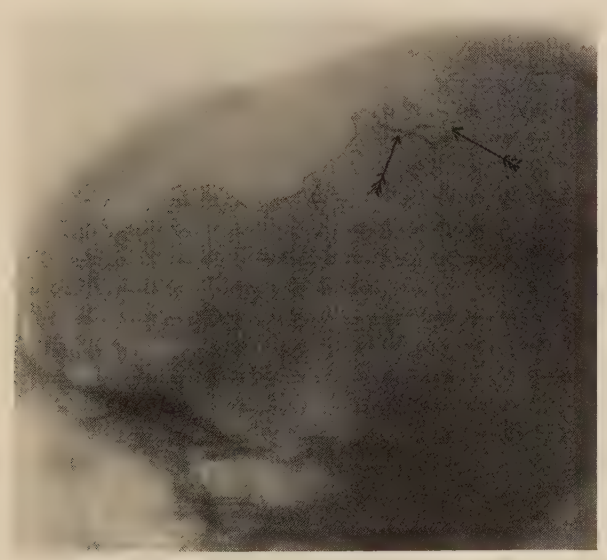


FIG. 16*A*.—Case IV*A*. Shows area of involvement before an attempt was made to cut around it.

This is a rather formidable array of facts against which to argue, and his conclusions, which have had wide acceptance, especially among the rhinologists, are in harmony with his findings.

We have been unable to substantiate all of his findings by referring to the literature.

Four of his cases were definitely described as having had radical treatment. But, in going over the cases, we find that:

B *C*

FIG. 16*B* and *C*.—Case IV*A*. Show the wide extension of bone involvement following an attempt to cure the patient by cutting out the diseased bone.

One described by Luc was still active after ten months. McKenzie himself, in a discussion of a case report of Mollison, said that at least six months should be allowed to pass without any symptoms before deciding that the case is cured, and other observers put the period as high as eighteen months.

Downey's recovery case was a chronic one of six years' duration, there was not a very widespread necrosis, and further it was said to have been a syphilitic case.

Syphilitic cases should not be recorded with these cases, as McKenzie himself said in throwing out one reported cured by conservative methods by Spicer.

Davis' case, quoted by McKenzie, seems to have been a postoperative case and to have recovered with very conservative treatment.

McCoy, 1910, reported a postoperative case that recovered with fairly conservative treatment.

Coffin, 1908, reported a case as cured of the osteomyelitis by operation, but also stated that the patient died. It is not clear to us whether McKenzie reported this as cured or not.

Harrison, 1912, reported a case of mastoid origin as having recovered with very conservative treatment and emphasized the importance of waiting for sequestra to form, of supporting the patient in general, and of using vaccines.

In our study of the literature appearing after these articles of McKenzie's; we found 25 cases reported, 12 following sinus operations, 12 arising spontaneously during the course of a sinus infection, and 1 after trauma.

We have had 6 cases of this type, 5 arising from the frontal, and 1 from the sphenoid sinus. This series we consider rather small upon which to base sweeping conclusions, but of those who have so vigorously advocated early radical treatment no one had more than 4 cases.

Tilley[2] reported 4 cases, all treated radically; 1 lived and 3 died.

Kernan[10] reported 2 cases; 1 treated conservatively that lived, and 1 treated radically that died.

Porter[11] reported 1 case following a sinus operation treated radically that lived for one year and then died of pneumonia. This is said to be the first postoperative case to recover.

Mollison[12] reported 1 case treated radically that lived, but at the time of the report not enough time had elapsed to be sure of a cure.

McClay[13] had 3 cases, 1 living, but no details of the treatment were found; 1 living following multiple operations over a period of six months or more; 1 dead two months after a nasal operation without further bone-cutting operation.

Skillern[14] reported 1 case that died after radical treatment, but concluded that radical treatment was delayed too long.

Bryan[15] reported 1 case, dead after removal of involved bone with curet, and 1 case living after ten or twelve months.

Milligan and Wrigley[16] gave one very short report of a patient recovering after radical operation.

McArthur[17] reported 1 fatal case without operation, but concluded that radical treatment was the best. However, he also stated that his case was too far advanced with involvement of bone in the ethmoid region for any treatment to be of avail.

Warren,[18] 1 case, reported living after bone-cutting operation, but reported after only about six months of observation.

Wood[19] reported 2 cases, with death of both following radical treatment; states that operations were too limited and too long delayed.

Bulson,[20] 1 case living following reported radical treatment. The operation, however, was two and a half months after the development of the disease, a time at which the diseased bone might have begun to separate spontaneously.

Eckstein[21] reported a fulminating case that died on the same day of radical operation.

Coffin,[22] as cited in previous footnote.

Turner,[23] 1 case, died after radical operation, and the author concluded that conservative treatment was best.

Van Den Wildenberg,[24] 1 case, death following radical treatment.

Von Eicken,[25] 2 cases, 1 dead following radical treatment, 1 dead from brain abscess, without much radical surgery.

Wolff Freudenthal,[26] 1 case, reported well six weeks after radical treatment.

McKenzie,[9] 1 case of limited necrosis, bone removed, death.

Patterson,[27] 1 case living after 12 operations of undetermined nature.

Tod,[28] 1 mastoid case, dead after radical operation on involved bone.

Knapp,[29] 1 case, dead after fairly radical surgery.

Thompson,[30] 2 cases, death of both following operations. Concluded that no kind of operations would stop the process, and mentioned 1 case that died after eighteen months.

McCoy,[31] 2 cases, 1 dead after radical treatment, 1 living after fairly conservative treatment.

Not all observers report favorably to the radical bone-cutting plan of treatment.

Harrison[33] reported the case described in preceding footnote.

Thompson,[30] as recorded here, concluded that the most vigorous treatment would not stop the process.

Lemere,[33] reported a case treated conservatively that died, but his conclusions as to the treatment of choice are not clear to us.

Kernan[10] leans somewhat to the conservative side, at least for chronic cases.

Opdyke[24] reported 1 postoperative case living after what seems to have been conservative treatment.

Lynah[35] reported 1 living with conservative treatment, but too soon to be sure of.

Turner[23] turned to the conservative side after losing a case treated radically, and said, "Once an osteomyelitis of the frontal bone is set up, surgical cure is practically hopeless."

Symonds'[36] case, in 1910, appears to have been treated by sequestrectomy.

Mosher, H. P., in a discussion of a paper by Bulson, sounds the same warning of conservatism that is attempted in this paper. He bases his views on observations of a case in which the osteomyelitis progressed after every radical surgical attack, but cleared up entirely under conservative treatment.

In the literature before 1899, at which time cases were first recognized as coming from the sinuses (except the mastoid), there are many reports and discourses on osteomyelitis of the skull bones. After discarding those definitely thought to be tuberculosis or syphilis of bone, some of the others must have been of sinus origin, though unrecognized as such.

Hewitt,[3] in the *Lancet*, 1864, has a long discourse on disease and necrosis of the skull bones, and emphasizes that loosening of the dead bone should be waited for and, except in cases with very limited involvement, radical surgery should never be done. The main object should be to support the patient.

Smith,[4] 1870, reported a case of spontaneous origin and separation.

Gardner,[5] 1865, reported a case with recovery following multiple sequestrectomies over a long period.

Gelston's,[6] 1847, case had full-thickness necrosis and died of meningitis after it was opened.

Hill,[7] 1843, had a long-drawn-out case of supposedly traumatic origin that was free of symptoms after a widespread operation twenty-five years after onset.

Marks,[8] 1894, reported a case during conservative treatment, but it was still slightly active at the time of the report.

Of our 6 cases:

1A.—One spontaneous frontal sinus case treated by late removal of the exfoliated bone survived and has been well for the past nine years, except for epilepsy, which is not related to the sinus trouble. (Fig. 14.) Patient referred by W. E. Sauer.

2A.—One frontal-sinus postoperative case had twenty-one drainages or removals of exfoliated, bone, died of thrombosis of an emissary vein after subsidence of all local symptoms and he had been sent home. (Fig. 15.) Patient referred by V. V. Wood.

3A.—One spontaneous frontal-sinus case died of purulent meningitis after conservative treatment for three months by her family physician. Referred by J. M. McClelland.

4A.—One frontal-sinus case treated for eleven months by 10 operations (the second one radical, and followed by further spread of the disease) and five blood transfusions. When he became so exhausted that further surgery was not to be considered, he was put on salvarsan (Wassermann negative) and milk injections, and improved so rapidly that he left the hospital in a month and has been working on his farm for the past five years. (Fig. 16, *A*, *B*, *C*.) Referred by W. E. Sauer.

5A.—One frontal-sinus case treated conservatively, had infections in two other parts of the body that may have been metastatic, but at present his head has healed and has been so for about a year. This case is still under observation and is not yet considered cured. Referred by Birsner.

6A.—One sphenoid case (none others found in the literature, except some of direct perforation of the bone), who died of meningitis with widespread necrosis of the base of the skull. Actinomycosis was found in conjunction with real pus. Referred by G. H. Copher.

While we had but 2 cases to survive out of 5 that we were able to follow to a conclusion, we are not yet ready to accept the more modern teaching of early, radical bone removal for these cases, and, until more conclusive evidence is adduced, will continue to apply the older rules of hygiene, drainage and the removal of exfoliated bone. To these we would add an attempt to raise the patient's resistance to an apparently low-grade infection that seems to cause insufficient tissue reaction to expel it, but which, like the rats we tolerate in our back yards, may be very deadly to the complacent host. Later, we may be able to make some report on the type of infection, and now have some observations. In the future we will add general quartz-light treatment.

Analysis of Jaw Cases

Number of Cases: 18 Children, 21 Adults (11 Male, 10 Female)

Cause	Apparently associated with peridental infections, 33. 31 of these were associated with extraction during acute stage of a peridental infection. Of these 31 acute infections, there were 2 following devitalization of the pulp with arsenic, and 1 following application of phenol to the pulp chamber 1 associated with salivation, HgCl. 1 associated with extraction of an old root.
Other causes than teeth, 6	1 associated with gland enlargement and loosening of teeth. 1 following tonsillitis. 1 following measles. 1 following diphtheria. 1 associated with tuberculosis elsewhere in the body. 1 associated with upper respiratory infection in an infant.
Jaws affected	Upper, 8. Lower, 32 (both jaws in 1 case of suspected syphilis).
Wassermann	Negative, 17. No report, 18. Positive or partly positive, 4.
Salvarsan	8 cases. 3 with negative Wassermann, improved. 3 with negative Wassermann, but no note of improvement. 1 with positive Wassermann, but no note of improvement. 1 with positive Wassermann, but treated before jaw was operated on. (2 cases with positive Wassermann had HgCl —2 and K.I. treatment only.)

Time elapsed between the appearance of first symptoms and the spontaneous throwing off, or the operative removal of sequestra in various cases. A total of 63 sequestrectomies, 8 of which were spontaneous.	1 at one week, bone-cutting operation on upper jaw—death. 1 at three weeks, spontaneous separation, upper jaw. 2 at one month. 2 at two months. 8 at three months. 7 at four months. 8 at five months. 8 at six months. 8 at seven months. 1 at eight months. 5 at nine months. 3 at ten months. 2 at eleven months. 1 at twelve months. 1 at seventeen months. 1 at twenty-four months. 2 at thirty months. 1 at thirty-four months. 1 at eleven years.
Number of operations . . .	25 cases—only one operation was necessary. 14 cases—multiple operations, but about half of these were external drainages.
Deaths	1 baby, following a bone-cutting operation on the upper jaw one week after the onset of symptoms.

Analysis of Osteomyelitis of Skull Cases

Authors, 33.		Number of cases with available records, 46 (2 mastoids).			
Radical	23	Radical treatment	37	Conservative treatment	9
Conservative	9	Dead	25	Dead	3
Undetermined	1	Living	12	Living	6
		Of the 12 living, 5 were reported too soon to be sure of, and in 1 there was a question as to whether the treatment was of a radical kind or not		Of the 6 living cases, 1 was a mastoid, 1 was a fairly limited case, and 1 was reported too soon	

Age.		Wassermann.	Vaccines used in:
Up to 10	6	Negative 16 Positive 0	5 cases of those that recovered with radical operation
10 to 20	9	Antisyphilitic treatment	
20 to 30	18		
30 to 40	10		
40 to 50	2	9 without results	2 cases of death with radical operation
60	1	1 with vaccine and radical operation, recovery	
Male 28	Female 28	1 with sequestrectomies but reported too soon	1 with sequestrectomies with recovery (mastoid case)

Bacteriology; reported here as designated in reports.		Cause of death.
Staph. 3	Proteus-staph.-short chain strep. 1	Basilar meningitis 1
Strep. 5	Mixed with strep. in short chains 1	Meningitis 11
Staph. aureus . 5	Strep.-pneumococcus . 1	Brain abscess 12
Staph. albus. . 2	Staph.-Gram-neg. bac. . 1	Throm. sup. long. sinus with meningitis 1
Short strep. . . 1	Staph.-albus-strep. . . 1	Pyemia with lung involvement 1
Nonhomo. strep. 1	Strep.-Gram-ps. diplococcus 1	Pyemia with meningitis . . 1
Enterococcus . 1	Staph.-strep. 2	Cavernous sinus thrombosis . 1
Actinomycosis . 1		Septic embolism of lung . . 1
Pneumococcus . 1		Lat. sinus throm. with meningitis (mastoid case) . . . 1

Cases Since 1913–1925

Origin	Frontal, 19; frontals and others, 4; antral-frontal, 2; spontaneous, 12; postoperative, 12; trauma, 1
Radical treatment .	Dead, 11; living, 8; 3 reported too early; 1 operated on after two and a half months' time
Conservative treat-treatment . . .	Dead, 2; living, 4; 1 reported too early
Osteomyelitis recognized . . .	Before operation . . . . . 21 At time of operation . . . . . 2 No note . . . . . 1 Outer table uninvolved and extent of inner table involvement missed 1

Analysis Cases Observed Here

Age	10, 10, 17, 20, 20, 33
Sex	Male, 3; female, 3
Wassermann . . .	Negative, 4; no report, 2
Salvarsan treatment .	3 cases—2 died, 1 lived
Bacteriology . . .	Strep., 2; short chain strep., 1; strep.-staph., 1; actinomycosis with a pus producer, 1
Origin	Spontaneous, 4; postoperative, 2
Sinus of origin . .	Frontal, 5; sphenoid, 1
Treatment and results	Conservative in 6 cases; 3 dead 2 cured; there was some bone-cutting done in one of these that was immediately followed by spread of the disease 1 still living after one year with head healed, but too early to call a cure

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DISCUSSION

Dr. James Edwin Thompson, Galveston, Texas:

I can fully substantiate Dr. Blair's remarks in every respect. Some of the serious catastrophes of my surgical career have been in removing inflamed bone, or rather of operating on it too soon. Those results stare me in the face, and occasionally when I go around the museum I see pathologic specimens that I am not proud of. It is not pleasant to see portions of a jaw with periosteal bone attached and appreciate that one had operated on that bone too soon, and probably removed two or three or four times as much as one had a right to.

Dr. Blair's last remarks about the peculiar behavior of osteomyelitis of the skull bones interested me tremendously, because I have recently seen one of these cases that was one of the most distressing in my professional experience. A child of about thirteen years of age was brought to me from the northern part of the state about a month after an operation for a supposed infection of the middle ear. She showed marked necrosis of the whole of the squamous portion of the temporal bone and contiguous parts of the frontal bone and parietal. There was loss of skin and scalp tissue, and the bone was exposed over a large area. She was quite sick and deeply anemic. I operated and removed all the bone that was frankly dead. I was never quite certain when I was in perfectly healthy bone.

I expected the case to get better and heal up right away. To my amazement it did nothing of the kind; it spread slowly in a most indolent way. Granulation tissue would form and a probe could be passed under it for quite a distance in contact with dead bone. I packed it and it would not drain properly. On and on it progressed to the occipital bone behind and across the skull to the other side. I operated a second and third time without stopping the process. Eventually we stopped operating.

We never found any organisms but staphylococci in this particular case. We transfused the child on several occasions. For some reason the process would not stop. The child eventually died of septicemia about seven months after the first operation. All we have to show for it is a beautiful mosaic of portions of skull bone that are exposed in the museum for any one to look at and to cogitate over. I have never seen anything like it. I hope I shall never see anything like it again.

Dr. Blair (in closing): I will just take time to give our statistics on mortality, because they are not brilliant at all. The first case we had was a child of about ten years of age which we treated in a conservative

fashion. We removed the sequestrum and the child got well. The second was this case I referred to, in which we operated ten times and then stopped operating and the child got well. The third was operated nine times, but all were drainage type of operations.

The infection was apparently mild. That cleared up, but the boy died sometime later from the infection, which traveled through an emissary vein, causing thrombosis and meningitis.

Another case was that of a negro girl who had had a number of drainage operations. We failed to find the pus. We were like the man who tried to shoo off the skunk; we were gentle about it because we did not want to go through an infected area into clean tissue. She had about 8 ounces of thick yellow pus between the hemispheres at autopsy.

Another case came in of a boy who lived on a farm. He was told to go home and stay out in the sunshine. We have not again heard from him.

The final one was a boy who came in with a fistula in the side of his face. He also had fifth-nerve neuralgia. We did drainage operations and watched him for a long time, but never got any bacteriologic reports that told us anything. He finally died of intracranial complications. The autopsy findings were extensive necrosis and basal meningitis which rose from a sphenoidal infection; this is the only sphenoidal case we could find on record. There was an actinomycosis demonstrated at autopsy.

Of the 3 antral cases, 2 that were not operated on recovered and 1 which I energetically drained promptly died.

PROGRESSIVE GANGRENOUS INFECTION OF THE SKIN AND SUBCUTANEOUS TISSUES FOLLOWING OPERATION FOR ACUTE PERFORATIVE APPENDICITIS

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AND

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The object of this communication is to report the histories of two cases of extensive destruction of the skin and subcutaneous areolar tissues, from infections occurring in the operative wounds of cases of acute perforative appendicitis.

While all surgeons are familiar with the various types of wound infection following operations on septic conditions of the various abdominal viscera, necessitating contamination of the wound surfaces during the operative procedures or following the drainage of these septic foci, few such superficial infections give rise to serious apprehension where the intraperitoneal focus ceases to extend and progresses normally toward recovery.

In practically all such cases, it is the deep-seated primary intra-abdominal infective process which causes anxiety and not the superficial secondary stitch abscess or subcutaneous cellulitis, for these almost invariably are controlled by the simple procedure of removal of sutures and establishing drainage of the subcutaneous space.

In the cases which I am now reporting, the opposite of this condition prevailed, and persisted to such an extent as to give rise to the greatest anxiety.

The progress of this infection was not rapid, as in a postoperative erysipelas, a streptococcus cellulitis, or gas bacillus infection; nor was it associated with high fever and grave toxemia usually

associated with these acute conditions. It was, on the other hand, exceedingly slow, extending only 1 or 2 cm. in the course of a week or ten days, and with little or no fever or evidence of toxemia. This type of infection seemed singularly rebellious to any and all types of ordinary wound treatment. It was apparently uninfluenced by local incisions, drainage, irrigation, heat, cold, Carrel-Dakin technic, or by the employment of other chemical disinfecting agents. It was not benefited by sunlight, Alpine light, or other forms of radiant energy.

The process may perhaps best be described as a slowly advancing white subcutaneous slough. The skin lesion, occurring somewhat later, appeared at first as a deep red or purple superficial edema, which later slowly broke down into a necrotic mass resembling an untreated carbuncle. This in turn was surrounded by a zone of lighter colored erythema, which gradually faded out into the normal skin. At times the subcutaneous necrotic area and cutaneous margins would assume a dark gray, or even black color. Beneath the sloughing area, which varied considerably in extent, sometimes being limited to the upper third of the subcutaneous fat and at others extending well down to the deep aponeurosis, there was always a layer of granulation tissue, which seemed to be a definite but ineffectual effort of repair.

In both of my cases the process seemed to start at the upper part of the cutaneous wound, and gradually crept downward along both margins toward the site of the intraperitoneal drain opening. The actual site of the deep drainage cavity was apparently never involved.

Without dwelling further on the special wound features of this infection, which will best be shown in the accompanying illustrations, I will briefly report the clinical features of the two cases, which in history, sequence of events, and wound appearances seemed identical.

Case I.—H. B. T., aged thirty-two years, an apparently robust and healthy man of good habits, and without evidence of chronic infectious or degenerative disease, while on a visit to New York, experienced a sudden attack of acute abdominal pain associated with all the characteristic symptoms and signs of an acute fulminating appendicitis. As he was alone in a hotel he did not obtain expert advice for two days. At the time the writer first saw him in consultation with Dr. Wendell

Mitchell, he had a temperature of 103° F., pulse 90. The abdomen was moderately distended and tender in all four quadrants; the area of maximum tenderness being at or near McBurney's point. Muscular rigidity was noted over the right rectus and neighboring muscles. As his prostration and general appearance seemed to indicate a grave infection, which had already existed for upward of fifty hours, he was removed to the Roosevelt Hospital for immediate operation. On opening the abdomen, the appendix was found to be completely gangrenous, and surrounded by an abscess containing about 30 cc., of thin foul-smelling pus. As the gangrenous process extended to the cecum, after removal of the appendix the cecal opening was closed by a purse-string suture, and the point of closure reinforced by suturing a small appendix epiploica and a layer of omentum over the stump. A large cigarette drain was left *in situ*, and the peritoneum, muscles, and skin partly closed by layer suture. Although the anesthesia was satisfactory and the operation of short duration, the following day the temperature rose to 104° F., the pulse to 120 and the patient seemed gravely ill. There was, however, abundant drainage from the deep sinus, necessitating frequent changes of the dressings.

On the third day, the cigarette drain was removed and a rubber tube inserted, through which the wound was frequently irrigated and treated by the Carrel-Dakin method. The discharge from the deep cavity promptly diminished, soon ceased, and the sinus closed satisfactorily.

During this period the temperature gradually declined and the patient's condition improved; and at the end of a week, he seemed thoroughly convalescent and no further anxiety was entertained regarding his ultimate recovery.

On the seventh day after operation a peculiar deep purple edema was noted around the points of entrance of the three upper silkworm-gut sutures. These were removed, the cutaneous wound opened and packed with sterile gauze. Two or three days later, to our surprise, the purple areas had not subsided, but apparently had extended to the margin of the then freely open cutaneous wound. They resembled six small carbuncles with beginning necrosis of the epithelial layer. Also it was noted that the edges of the wound beneath these lesions was undermined and presented isolated areas of a white slough, which extended down to various levels in the subcutaneous fat. Incisions were later made into these areas of necrosis, and the entire wound packed loosely with gauze covering several Carrel-Dakin tubes, and was constantly treated with accurately prepared Dakin fluid. The original sloughs gradually separated, but beyond this loss of tissue there appeared an extending zone of the deep red edema, which formed an elliptical area of discolored

skin about the size of a human hand, surrounding the upper limit of the original incision and extending downward on each side of the now widely separated cutaneous margins, almost but not quite to the lower pole of the incision which was the original point of entrance to the deep drainage opening.

As it was evident that the Carrel-Dakin treatment failed to control the process, a number of other antiseptic agents were tried, including iodine, iodoform, various mercuric compounds, hot poultices, and finally exposure of the wound to *x*-rays, Alpine light, and the direct rays of the sun. None of them seemed to have the slightest effect on the slow progress of the disease, which gradually extended peripherally and sloughed centrally until there was an ulceration which extended from a point near the iliac crest half-way to the lower costal border, and well over toward the median line, making an oval sloughing ulcer about 15 to 16 cm. in length by 10 or 12 cm. in width.

During the three weeks which had elapsed since the operation, the abdominal sinus had closed, the patient was practically afebrile, had a fair appetite, and seemed constantly improving. The only major complaint being the intense pain which was occasioned by the dressings and any manipulation of the wound.

During the latter part of this period he was seen by perhaps six or eight members of the staff, including general surgeons, experts in dermatology, bacteriology, and internal medicine, none of whom had ever seen a similar lesion arising from an operative wound.

Numerous cultures were made from the tissues and wound secretions, as well as blood tests to exclude syphilis, tuberculosis, blastomycosis, and other rare forms of infection. The wound cultures contained a large variety of organisms, but led to no definite conclusions.

At last, as it seemed evident that unless something far more radical were done in the way of treatment, a large part of the integment of the abdominal wall would be destroyed, it was decided to give a general anesthetic and to circumscribe the entire diseased area, well beyond the lesion, by an incision through the skin and entire thickness of the subcutaneous fat to the sheath of the rectus and aponeurosis of the external oblique muscles. This was done, and the long elliptical incision packed with gauze wet with a 1 per cent solution of formalin. This was changed every day, at first under gas anesthesia, and the packing kept constantly wet with the same solution.

The sloughing process continued from the original edges of the ulcer toward the incision, but never passed this barrier; and when all the intervening tissues were destroyed, we had an extensive granulating surface with healthy edges, which quickly took on the normal process of repair. We hesitated to advise a skin graft on account of the doubt-

ful nature of the process, and a very definite fear on the part of all who had observed its ruthless progress, lest any such procedure might again favor its reappearance.

By simple dressings and sun exposures, cicatrization took place rapidly, and at the end of thirteen weeks the patient was discharged cured.

Shortly after the experience just related, there appeared in *Surgery, Gynecology and Obstetrics* the report of a similar case by Dr. Thomas S. Cullen of Baltimore, admirably illustrated by two colored plates of the lesion. From Dr. Cullen's description of the etiology, symptoms, progress and wound appearances, I was convinced that our two cases represented examples of a rare but definite type of wound infection, probably due to some organism derived from the intestinal tract, as both arose from the operative treatment of a perforated intestinal lesion.

I will not burden you with a detailed account of his case, as doubtless you all are familiar with his report; but will content myself with showing two lantern-slide photographs of the lesion, which he kindly sent me.

My second case occurred during the past winter.

Case II.—L. M., male, aged sixty-four years, but whose appearance and athletic vigor would give one the impression of a much younger man. On December 18 last, he was admitted to the Presbyterian Hospital for operation for acute appendicitis. The duration of his illness had been about thirty hours. On admission his temperature was 100.8° F., pulse 110; abdomen moderately distended, with marked tenderness and muscular rigidity over the appendix area.

On operation, the appendix was found to be partly gangrenous, perforated and surrounded by a collection of thin odorless seropus. The intestines in the immediate neighborhood were reddened and covered with fibrin. The appendix was removed, the wound partly closed, and a large cigarette drain introduced which extended to the region of the appendix stump. He reacted favorably to the operation, the abdominal symptoms subsided, the temperature and pulse improved; and his general condition at the end of three days seemed entirely satisfactory. On the fifth day, small areas of necrosis were observed around the points of entrance of the upper two deep silkworm-gut sutures. The stitches were removed, the wound edges separated, and the wound lightly packed with gauze. The Carrel-Dakin method was employed, which at first seemed to act favorably, as for four or five days the wound

showed no further changes. It did not then occur to the writer, that we had to do with other than a trivial secondary infection of the superficial wound. The deep drain was removed on the sixth day, a small rubber tube substituted, and the deep wound frequently irrigated. About the tenth day, after the deep drainage sinus had ceased discharging, and the lower part of the wound seemed normally granulating, the same deep red discoloration and edema, as in Case I, appeared around the upper part of the wound, with four or five oval elevated lesions at the site of the stitch openings. There was also noted a subcutaneous area of white slough, which extended from the edges of the wound upward beneath the unbroken but edematous skin, toward the stitch openings. A day or two later, two or three of these necrotic areas were removed, leaving an irregular undermined unhealthy looking skin border. At this time also it was noted that the deep purple edematous nodules showed signs of breaking down, and that the area of hyperemia was extending outward into the surrounding skin. One or two black areas were also seen along the skin margins. It was then recognized for the first time, that we had to do with the same type of spreading gangrenous cellulitis and dermatitis as in the first case.

On January 9, under gas-oxygen anesthesia, the entire cutaneous margin of the wound was excised, including all of the reddened and edematous tissues, the wound treated with peroxide of hydrogen, douched with sterile water, and packed with gauze soaked in and kept moist with Dakin solution. The process seemed arrested for two or three days; but later the same characteristic deep purple lesions occurred along the cutaneous borders, and sloughing of the fatty tissue was apparent. Both the red edema and the subcutaneous necrosis spread more rapidly than at first, in spite of the most careful Carrel-Dakin technic; and the patient showed a definite increase in temperature and pulse-rate (Plate A).

We were then convinced that the first excision had not been sufficiently wide to insure removal of the deeply imbedded organisms, which evidently had penetrated into the tissues well beyond the outermost limits of the hyperemia and edema. Again under gas-oxygen anesthesia on January 18, a wider excision was made from 3 to 4 cm. from the wound edge, this time removing the entire infected area and surrounding skin. The floor of the large wound thus created was treated by peroxide of hydrogen, douched with sterile water, and packed with 1 per cent wet formalin gauze as in the first case. This was kept in place for two days, and constantly wet with the dilute formalin solution. The formalin was then discontinued and the regular Carrel-Dakin treatment carried out. This served to arrest the process, no further characteristic lesions appeared on the skin edges, and the entire wound took on a healthy granulating appearance.

The patient was discharged from the hospital at the end of seven weeks, and later at his home continued treatment by exposure to the sun, and the use of other stimulating measures.

Dating from the last operation, the time required for healing this extensive wound was about eleven weeks. It occurred without the aid of skin grafts.

As in my first case, the one most distressing symptom was the acute sensitiveness of the wound, making dressings and all manipulations about the lesion extremely painful. It is worthy of note also, that as soon as the specific infection was killed, this sensitiveness promptly disappeared.

Since the observation of my second case, four other similar cases have been brought to my attention.

The first of these was a woman, aged sixty-three years, who was admitted to the Presbyterian Hospital in January, 1919, for observation. Four or five months before her admission, what was thought to be a cold abscess was present in the upper right quadrant of the abdomen. This was opened and drained. Shortly after the operation, there occurred a slowly spreading gangrenous ulceration of the skin, beginning at the wound edges, and extending in all directions, until, at the time of her admission to the hospital, the ulcerated area occupied a large part of the upper half of the abdomen, and extending well down into the right flank.

The process had been exceedingly slow, and at first was thought to be due to syphilis, tuberculosis, or one of the mycotic infections. Each of these however was excluded by careful tissue examinations, bacterial cultures, and inoculation experiments.

The cultures from the tissues and wound secretions showed a large variety of organisms, but no definite conclusion was reached.

Although the origin of the original abscess, whether mural or intra-abdominal and possibly due to a small perforative lesion, could not be ascertained, Dr. Whipple, who saw the case several times in consultation, informed me that it was similar in many respects to my second case.

The second was one reported at the joint meeting of the New York and the Philadelphia Surgical Societies during the past winter by Dr. E. G. Alexander. His case also followed operation on an acute perforative appendicitis. The progress of the case was similar to the two reported in this paper, and although he strongly advised complete excision of the area, the patient refused operative treatment, with the result that the ulceration continued to extend until it involved a large part of the right

half of the abdominal wall, the right buttock and thigh to the knee. His patient was in the hospital eleven months.

The other two cases were one reported by Dr. Frederick Christopher, of Chicago, and another which occurred in the service of Dr. A. Moschcowitz, of New York.

As soon as I recognized that the process in my second case was the same as in my first patient, I called in consultation Dr. Frank Meleney, of the surgical staff of the Presbyterian Hospital, with a view to obtaining his advice and coöperation in determining the causative organism or organisms. He observed the patient from time to time, and entered enthusiastically into the plan of establishing the bacteriology of the process.

The report of his observations and inoculation experiments is as follows.

Bacteriological and Experimental Study. The fluid from the peritoneal abscess was cultured in the usual way by a member of the laboratory staff. A pure culture of nonhemolytic streptococcus was reported. No anaërobic cultures were made and it is not on record whether or not the culture grew in the usual manner on the surface of a blood-agar plate. It therefore remains an open question whether this organism was or was not the same streptococcus which is to be described below. Later, when the gangrene developed in the wound, a second routine aërobic culture revealed a hemolytic Staphylococcus aureus and a diphtheroid bacillus. The streptococcus was not found.

Subsequently a special study was made of the slough from the wound. A hemolytic Staphylococcus aureus and a diphtheroid bacillus were found again aërobically, while the anaërobic blood-agar plates revealed a streptococcus which would not grow on the aërobic plates.

When the region was prepared for complete excision, the surface was painted copiously with iodine in order to minimize surface contamination. A culture was taken of the blood drawn by the first incision (at point 1 in Fig. 1). After excision, the complete specimen was taken in a sterile towel to the laboratory, the surface was again painted with iodine and incisions were made just beyond the gangrenous margins of the ulcer at points 2, 3, 4 and 5 in Fig. 1. Direct smears of the tissue fluid in these regions showed, in the two nearest the margin, great numbers of very small cocci

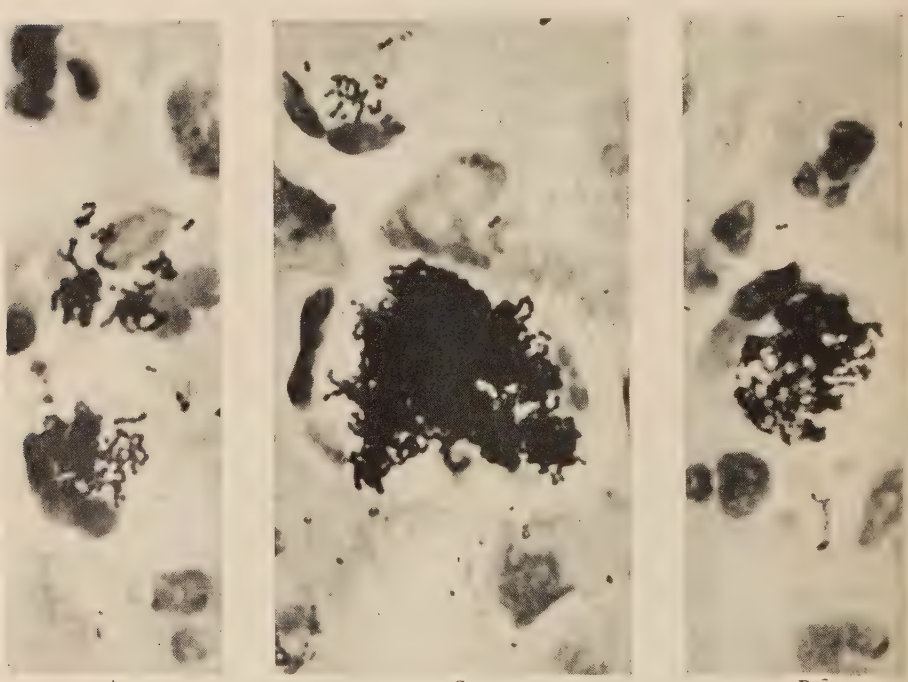
PLATE A.—Appearance of the wound before the second excision; the lower granulating portion of the incision being drawn together by adhesive plaster.

growing in masses of tangled chains. Many of these were found to be contained in leukocytes (Fig. 2, *a*), some stretching the cell

FIG. 1.—The excised lesion after formalin fixation. The figures indicate where cultures were taken.

membrane almost to the bursting point, (Fig. 2, *b*), and others in masses much larger than the phagocytes with cellular debris in the neighborhood, (Fig. 2, *c*). These findings seemed to represent

different stages in the battle between the foreign invaders and the host, with apparent demonstration that the organisms were not only growing in the tissues but in the phagocytes as well. In the two regions farther from the margins, diplococci and short chains were found both within and without the tissue cells and wandering cells. Sections of the tissue stained for bacteria revealed them in great numbers (Fig. 3). Cultures were made from the five points



A C B

FIG. 2.—*A*, phagocytes containing a few chains of cocci. *B*, a phagocyte distended with organisms. *C*, Tangled chains of organisms with cellular fragments in the neighborhood. Gram stain. Oil immersion lens.

mentioned above, aërobically and anaërobically on 5 per cent sheep-blood-agar plates and in cooked-meat medium with and without dextrose. In twenty-four hours three out of four of the cultures yielded, in pure culture, a nonhemolytic streptococcus which grew in the cooked-meat medium with or without dextrose, both aërobically and anaërobically. On the blood-agar plates, however, it failed to grow except under anaërobic conditions, the

aërobic plates being without any evidence of growth. The fourth culture showed beside the streptococcus a few colonies of non-hemolytic Staphylococcus albus on the aërobic plate, possibly an air contamination. After forty-eight hours the culture of the blood from the incision revealed a pure culture of the same streptococcus. Thus in four out of five cultures from regions extending

FIG. 3.—Organisms in tissue section. Gram stain. Oil immersion lens.

from just outside the margin of the ulcer to the limits of excision, this organism was found in pure culture, evidently growing in the tissues and successfully combatting the defensive efforts of the host. The evidence seemed to be strong that this bacterium was in some way related to the disease process. Contrary to expectation, the hemolytic Staphylococcus aureus and the diphtheroid bacillus cultured from the slough, were not found in the zone

outside of the gangrenous margin. This we considered to be most significant because it indicated that for the infection as a whole the streptococcus was the more important. Experiments were then carried out in order to determine the cultural characteristics and the pathogenicity for animals of this micro-aërophilic nonhemolytic streptococcus.

Cultural Characteristics

It is a very small coccus growing in chains of varying lengths. The individuals are approximately 0.2 to 0.5 microns in diameter. It is Gram-positive. It grows readily in the anaërobic jar in cooked-meat medium with 0.2 per cent dextrose. In this medium it tends to form tangled chains in a manner similar to its growth in the tissues. It develops much less readily aërobically in the cooked-meat medium without dextrose. Anaërobically on 5 per cent sheep-blood-agar plates in twenty-four hours it grows with small pin-point nonhemolytic colonies which become slightly green on standing in the air. (Aërobically it would not grow on the blood-agar plates until it had been passed through nine aërobic subcultures in cooked-meat medium. It then very slowly developed minute colonies. This ninth subculture was sealed and kept for a month in the ice-box. It was then found to have lost its ability to grow aërobically on the blood plates.) When grown anaërobically this organism will ferment dextrose, lactose, saccharose and salacin, but not mannite. It is killed in fifteen minutes when heated to 60° C. The organism may, therefore, be classified as a nonhemolytic micro-aërophilic streptococcus, similar in many respects to certain of the intestinal streptococci. The intestinal origin of the streptococcus in this case is not proven; it is only presumptive.

Animal Inoculation

Two cubic centimeters of a twenty-hour culture in cooked-meat medium with 0.2 per cent dextrose injected into the peritoneum of a mouse failed to kill. Five cubic centimeters in the peritoneum of a guinea-pig failed to make the pig ill but the organism was recovered from the peritoneum after two weeks and, in another instance, a month later. When the ninth subculture mentioned

above had acquired the faculty of growing on aërobic plates, it was injected into the peritoneum of a guinea-pig. It was recovered after twenty-four hours and was then found to have lost the faculty for aërobic growth. Five cubic centimeters subcutaneously in guinea-pigs and rabbits produced slight redness and swelling which appeared on the day after injection, but rapidly subsided. There was never any evidence of gangrene. These failures to demonstrate pathogenicity for animals seemed to controvert the very definite evidence of activity in the tissue of the patient. It was then suggested to try the effect of this organism combined with the hemolytic Staphylococcus aureus and the diphtheroid bacillus with which it was found associated in the actual gangrenous margin of the wound. Two cubic centimeters of a twenty-hour culture of each organism were injected subcutaneously into control guinea-pigs and into another pig 1 cc. of the hemolytic Staphylococcus aureus was combined with the same quantity of the nonhemolytic micro-aërophilic streptococcus. In still another pig the streptococcus was combined with the diphtheroid bacillus. In twenty-four hours the staphylococcus combined with the streptococcus had produced a large, red, tender swelling 2 by 3 cc. in diameter with a central area of discoloration indicating beginning gangrene. The staphylococcus alone produced a somewhat smaller, red, tender swelling, without any evidence of gangrene. The streptococcus alone produced only a slight red swelling, as did the combination of streptococcus and diphtheroid bacillus. The diphtheroid bacillus alone produced no lesion.

In the second twenty-four hours the lesion with the staphylococcus alone increased slightly in size but thereafter subsided, finally localizing as a small abscess from which the organism was recovered after two weeks. The lesion produced by the combination of staphylococcus and streptococcus increased in the second twenty-four hours and a large irregular area of frank gangrene developed which separated at the margin after five days and sloughed out. Both organisms were recovered from the lesion. This experiment was repeated twelve times in guinea-pigs and rabbits and in every case but one produced a large lesion with more or less gangrene. The lesion in one of the guinea-pigs is shown in Figs. 7 and 8. In one rabbit the mixture produced no gangrene but an abscess formed three times the size of the controls. In one instance a silkworm-gut suture contaminated with the

staphylococcus was passed through an area into which the streptococcus had been injected and it was tied with moderate tension.

FIG. 4.—The lesion in a dog, as it appeared on the fourth day. On the left side of the picture is the swelling produced by the staphylococcus alone. On the right is the swelling produced by the streptococcus alone. In the center is the lesion produced by the combination, showing early gangrene. One-half life size. Slightly reduced

Gangrene developed around the suture, with a wide zone of swelling outside of it. The process generally reached its peak in three to

four days, and thereafter subsided. It did not spread progressively as in the human case. On 3 occasions out of 9 the staphylococcus, when injected alone in double doses superficially in thin-

FIG. 5.—The lesion in the same dog on the sixth day. One-half size slightly reduced.

skinned pigs, produced some superficial gangrene, but in 6 cases it simply formed a small abscess. Because of this occasional inconsistency, the experiment was repeated in a dog in order to use an animal with thicker skin and more subcutaneous tissue.

On the right side the staphylococcus was injected alone, on the left side the streptococcus alone and in the center the same quantity of these two, mixed together. A total volume of 5 cc. was injected into each area, the pure cultures being diluted with broth. Care was taken to inject into the subcutaneous tissues with a minimum of trauma at the point of injection. In twenty-four hours a moderate swelling appeared at the site of the staphylococcus injection and a slightly larger one at the site of the combination. Only slight swelling appeared where the streptococcus was injected

FIG. 6.—The lesion in the same dog on the seventh day, showing undermining. Life size. Slightly reduced.

alone. In four days the mixture had produced a large swelling four times as large as the staphylococcus alone and there was an irregular patch of early gangrene in the center. The staphylococcus lesion thereafter subsided without gangrene.

On the fifth day frank gangrene developed at the injection site of the mixture and on the sixth day it sloughed out leaving an undermined gangrenous margin. This spread slightly for a day or two and then subsided but showed very little tendency to heal. The dog died of pneumonia on the eighteenth day after inoculation

Stages of this process are shown in Figs. 4, 5 and 6. The similarity of Figs. 1, 6 and 8 is striking.

FIG. 7.—The lesion produced in a guinea-pig by a mixture of the streptococcus and the staphylococcus after forty-eight hours. The light areas in the center are yellow dead skin. Life size. Slightly reduced.

COMMENT. Although no definite conclusions can be drawn from these experiments, there seems to be evidence that in guinea-pigs and in rabbits and in the only dog in which the experiment was tried the nonhemolytic micro-aërophilic streptococcus and

the hemolytic Staphylococcus aureus cultured from the patient's wound, when injected together, produced gangrene almost invariably, while these organisms injected separately did not do so—the

FIG. 8.—The lesion in the same guinea-pig after six days. There has been a slight spread involving the umbilicus. Life size. Slightly reduced.

streptococcus never and the staphylococcus only rarely in thin-skinned guinea-pigs. These experiments suggest that a certain symbiotic function or a combination of functions of these two organisms may be necessary for the production of the gangrene.

The rarety of its occurrence clinically may be explained by the necessity for the coincidence of two such organisms in the wound. The streptococcus is much more rare in its occurrence than is the Staphylococcus aureus and the streptococcus might easily be overlooked in routine cultures.

Which organism is the essential one in the production of gangrene is a question not yet fully answered. In this patient the streptococcus was the one more extensively invading the tissues and the staphylococcus was only present in the gangrenous margin. When the gangrenous margin was partially excised, the process continued to spread, but when it was completely removed, the process ceased. Even though the cut margin yielded a growth of the streptococcus, the body took care of the infection which remained. In animals the staphylococcus produced the greater lesion when injected alone, but on one occasion a mixture of this streptococcus with a control hemolytic Staphylococcus aureus produced an extensive gangrenous lesion, while the staphylococcus from this case mixed with the diphtheroid bacillus produced no gangrene. We favor the following explanation for the phenomenon: The infection with the streptococcus prepared the way for the gangrenous process, but for its actual production a second organism was necessary, in this case the Staphylococcus aureus.

The solution of this problem will require more extensive experimentation, but the reaction of laboratory animals to infection is often so different from that of human beings that it will be well to avail ourselves of every opportunity to study this condition as it exists in man. These cases are so uncommon that it is not likely that one person would have the opportunity to study such a condition more than once or twice. Our findings are reported in detail in order that they may be compared with similar studies made by other observers. It will be of interest to search for such a combination of organisms in other lesions of this kind, for it is only by repeatedly finding these conditions prevailing that we can be certain that the cause of the disease has been demonstrated. It is not unlikely that such combinations were present in the cases previously reported by Cullen, of Baltimore, Christopher, of Chicago, Alexander, of Philadelphia, and in the case observed by Moschcowitz, of New York, and in the first case seen by Dr. Brewer.

DISCUSSION

Dr. Charles A. Porter, Boston, Mass.:

I can recall 3 similar cases at the Massachusetts General Hospital—2 followed appendicitis and 1 an operation for gangrenous gall-bladder. The woman, about thirty-five years of age, with the gangrenous gall-bladder, had a rather more chronic process than this; a slow and steady sloughing of almost all of the skin of the abdomen and chest, except the two nipples, which remained normal.

These 3 cases were investigated in every ordinary way, both bacteriologically and histologically, but not as carefully as Dr. Brewer's, and we could draw no conclusion. They were all cured ultimately by radical excision and skin-grafting.

Dr. Alexis V. Moschcowitz, New York City:

I wish to congratulate Dr. Brewer upon his success, not only in the therapy, but also in his search for the infecting organism of this apparently rare malady. I have had under my observation 2 cases of this nature. My first one was extremely fulminating; the second was of a more or less chronic nature, of which Dr. Brewer's case was typical. The first case resulted from a trivial trauma in the left temporal region. When I first saw the case it resembled a small carbuncle with a black, necrotic, central area. Under our eyes this necrotic area spread further and further, until it involved practically the entire half of the head, face and neck down to the clavicle. We tried the gamut of all the antiseptics and finally resorted to the actual cautery; however, without any material improvement in the lesion and the patient died. I have never seen a case of so-called "hospital gangrene" of the older writers, but upon consulting the literature appertaining to the subject, the case impressed me as such. The second followed an operation for acute suppurative appendicitis and was of a much more chronic nature. The progress was stayed by burning a groove into healthy tissue with the Paquelin cautery. In both cases we made repeated attempts to isolate the causative organism but without any result.

Dr. Marshall Clinton, Buffalo, N. Y.:

I should like to report a similar case to the ones Dr. Brewer presents. After a period of spreading gangrene for seven weeks the process extended from the pubes below to the costal margins above, on the left side, to the anterior axillary line and to the right, past the postaxillary line. We used every therapeutic measure we had ever heard of without success. At the suggestion of our director of laboratories, we applied a 10 per

cent solution of sodium chlorid to the margin of the ulceration, and within a period of six days the purplish-red, swollen edge subsided everywhere, leaving a clean, healthy, granulating ulcer. Followed skin-grafting and a quick convalescence.

Dr. Henry H. M. Lyle, New York City:

I should like to ask Dr. Brewer whether there is a relationship between this and the form Meneley has described as subcutaneous streptococcus gangrene. What makes me think of this possibility is that we had a typical subcutaneous streptococcus gangrene that started in the leg, and when it reached the abdomen its character changed. We would not have thought very much of this if we had not had a second case which behaved in exactly the same manner. When the process reached the abdomen its character changed exactly as in the first case. Can it be that the anatomic arrangement of the subcutaneous tissue and the blood supply of the abdomen alter the local picture?

Dr. Brewer (in closing):

I have been very much interested in knowing there were as many cases as have been reported today. These are the only 2 cases that have come under my observation.

I am impressed with what Dr. Bevan says, because they do resemble the old phagedenic chancres that I used to see in the venereal clinic thirty odd years ago. Also, there was a type of gangrene which they had in the Civil War which was called hospital gangrene, in which they used bromin.

These old army surgeons poured pure bromin from a bottle into the wounds and, although it probably had good effects on the bacteria, the patients rarely recovered. It never occurred to me to try bromin in these cases, because I have felt for a great many years that we have a very powerful bactericidal agent in a 1 per cent formalin solution, that is 1 per cent of the commercial formalin. I used it twenty-five years ago in the old City Hospital on Blackwell's Island, because it was the only agent I found that would control certain types of infection that we had in those very debilitated subjects, most of them having diabetes, Bright's disease, tuberculosis or syphilis. It occurred to me that in these cases, if we could circumscribe the entire area an inch or two away, and then pack with formalin, it would not pass that barrier, which it did not.

Of course, the surgical procedure was to make a complete excision in the more extensive cases. In my second case I did not at first use formalin; I used Carrel-Dakin, but the infection passed immediately beyond to the healthy tissues, and spread in every direction, even more

rapidly than before. In my second excision I went back to my old formalin. That apparently killed the infection.

One characteristic of both of these cases was that the wounds were excessively sensitive, any manipulation causing severe pain. You noted in both cases I had to do the subsequent dressing, for four or five times, under general anesthesia. Just as soon as the wound was free from that particularly type of infection it was no more sensitive than an ordinary wound.

I am sorry I could not read all of Dr. Meleney's report. My summary of it does not do justice to him. He has made a most careful bacteriologic investigation, and has given us a complete report, which will appear in the *Transactions*. So that if any of you encounter similar cases you may have the benefit of his observations.

THE ART AND SCIENCE OF PLASTIC SURGERY

By JOHN STAIGE DAVIS, M.D.
BALTIMORE, MD.

(From the Surgical Department of the Johns Hopkins University and Hospital)

Introduction. It is a significant fact and one which is very gratifying to me to have plastic surgery deemed of sufficient importance to be chosen by the American Surgical Association as the topic for a symposium, as this means that this branch of surgery has assumed its legitimate position.

The titles of the papers to be read in the symposium today give some indication of the scope of the work, but I feel that it may not be amiss for me to discuss the subject as a whole rather than take up any particular phase, as during the last twenty years I have been actively interested in the development of plastic surgery in this country and have possibly had something to do with placing it upon its present status as a definite subdivision of general surgery.

The science of plastic surgery is the organized knowledge of the fundamental principles involved in the transplantation and shifting of tissues and the clear understanding of what can be accomplished in this work. The art of plastic surgery is the application of this knowledge and the actual manipulative reconstruction. Bulwer-Lytton says that "art and science have their meeting-point in method," and this is especially true in plastic surgery.

What is plastic surgery? This is a question which is frequently asked and is almost as frequently incorrectly answered, as there is a great deal of misapprehension as to its scope and the possibilities of the work.

Plastic surgery is that branch of general surgery which is distinctly formative or restorative. It deals with the reconstruction of injured, deformed or lost parts all over the body, with the

reëstablishment of function and incidentally with the improvement of appearance. Is plastic surgery anything more than surgery of the skin and adjoining mucous membranes? Most emphatically, yes; in fact, a great deal of the work is of a major character. While in many instances, the lesions include only the skin and adjacent soft parts, frequently the deeper tissues are involved and often the supporting frame-work.

Development. There are still many who wrongly believe that plastic surgery was entirely a development of the World War and that nothing had ever been previously done in plastic or reconstructive work.

The truth is that plastic operations were described in the earliest Indian and Egyptian records. Furthermore, plastic work was apparently well known in the early part of the Christian era. Then for many years the art seems to have been lost, at least to European surgeons, until about the middle of the Fifteenth Century when a Sicilian surgeon is said to have restored a lost nose by using a flap from the arm.

The first book on plastic surgery was published in 1597 by Gaspar Tagliacozzi and in it he described several operations, but gave special prominence to his method of rhinoplasty in which he used a pedunculated flap from the arm. In the course of a few years this work was forgotten and it was not until the beginning of the Nineteenth Century that real interest was taken in plastic surgery by the foremost surgeons. During the first half of the Nineteenth Century great strides were made in evolving principles and working out operative procedures. Some of the most distinguished surgeons of that time are known today only by the plastic operations, which have their names. The transplantation of skin and other tissues was a development of the latter part of the century.

Most of the fundamental ideas on which are based the majority of the plastic operations used today were worked out during the Nineteenth Century, some of them more than one hundred years ago.

Before the World War such men as Morestin, Nélaton, Ombrédanne, put France, in plastic surgery, far ahead of other European nations. In Germany, Lexer was preëminent in plastic work and created much interest in the subject. In England, apparently, no one of note was interested; while in other European countries,

except for certain surgeons who specialized in operations on the nose, eyelids or lips and palate, there were no outstanding figures. In the United States every general surgeon was doing plastic work as it came along in his service. No one was specializing in this subject and none of the busy surgeons had time or inclination to delve deeper into it.

About twenty years ago, Dr. Finney, knowing of my interest in the subject, advised me to concentrate on this work. As I became more and more familiar with the scope of plastic surgery I found that the literature, which dated from the earliest times, was very widely scattered and that no attempt had been made to gather the threads together and coördinate the different branches of the subject. Moreover, it had apparently not occurred to anyone that the field was large enough or important enough to become a separate subdivision of general surgery. In fact, the idea of combining the plastic problems of the entire body, as far as I could find out, had not previously been thought of. I was speedily convinced of the importance of plastic surgery and soon saw the necessity of its development into a subdivision of general surgery. This work was well under way before the World War began.

Many of you are unaware of the bitter opposition which developed when the suggestion was brought forward that plastic surgery be made into a definite subdivision. It is an interesting story which I will not take up at this time. I am glad to say that at the Johns Hopkins Hospital the matter has finally been straightened out and plastic surgery is now on a firm basis.

There is no question but that the war stimulated general interest in the subject; but, as a matter of fact, little advance was made in the principles of plastic surgery, although the very large number of cases available made it possible to standardize and improve operative procedures in certain groups of facial injuries. With few exceptions, the group of men assigned to plastic work during the World War were those who had previously confined their practice to eye, ear, nose or throat surgery, or to dentistry, and few had had a general surgical training. The majority of them, I venture to say, had never done a plastic operation and were ignorant of the literature of the subject. In consequence, much time was wasted and much suffering was endured by the wounded; but gradually satisfactory methods were developed, which were based

largely on the elimination of procedures which had failed. Some of these methods have since been reported as new, which are either modifications of well-known procedures or rediscoveries of methods long familiar to the student of plastic surgery.

It is a well-recognized fact that few general surgeons can do plastic work well, so it is becoming more and more the custom to refer such cases to those especially trained along this line. If the best interest of the patient is taken into consideration, this is a logical move and is a development similar to what happened when genito-urinary surgery, orthopedic surgery and neurologic surgery were split from the general surgical tree. No one can gainsay that in these conditions better treatment is given patients by trained specialists in these subbranches of surgery than when they are looked after by the general surgical service, and this is particularly true when dealing with plastic cases.

There is no division of surgery which does not call on the plastic surgeon at one time or another as problems arise, and I have found that it is a much-needed development.

In a paper published several years ago I used the term, general plastic surgeon, to distinguish the few surgeons who have given special attention to plastic problems of the entire body from those who have confined their plastic work to facial reconstruction of one sort or another. So when I speak of a plastic surgeon I mean a general plastic surgeon, as I do not regard a man who confines his work, say, to noses or eyelids or palates, or even to the face alone, as fully qualified except in a limited field.

Scope. To the layman the face is the only field for this work, and, strange as it may seem, the same idea is often held by the profession. The limitation of plastic surgery during the World War, by military regulation, to maxillofacial reconstruction has undoubtedly had much to do with the erroneous and widespread impression that plastic surgery is entirely confined to the face. Unquestionably this is a very important branch of the subject, but plastic work on the trunk and extremities is equally important. The principles are the same, but the problems are entirely different. I should again like to emphasize the point that plastic surgery should be visualized as a whole and not be thought of from the viewpoint of anyone of its subdivisions.

The only way to educate the laity as to the possibilities of plastic surgery and to warn them of the danger of such things as the

injection of paraffin under the skin, which is so frequently done by cosmetic surgeons, is first to educate the medical profession so that they really know what true plastic surgery is. When this has been accomplished then the task of educating the public will be simpler.

PLASTIC VERSUS COSMETIC SURGERY. Plastic surgery is confused in the minds of many with the work done in beauty parlors. Without question, plastic or reconstructive surgery is absolutely distinct and separate from what is known as cosmetic or decorative surgery. As a matter of fact, a very small part of real plastic surgery is done for cosmetic or decorative reasons only. All plastic surgeons have to do cosmetic work occasionally, usually in the course of other procedures, but do any of you happen to know of a trained surgeon of standing in the profession who has become a decorative surgeon?

The so-called "plastic surgeons," who are usually well advertised, and who are really "beauty doctors," should be termed cosmetic or decorative surgeons, as few of them have even a glimmer of the principles of legitimate plastic surgery, and none of them are interested in the real problem of the work. When possible, I avoid even the use of the word cosmetic[1] in my reports, as I feel that it has little place in true plastic surgery, whose main object is the correction of actual deformities.

Personally, I have never cared to do cosmetic surgery, as its aims do not coincide with my conception of what true plastic surgery should be. I am sure that Dr. Lewis agrees with me when I say that at the Johns Hopkins Hospital we are not interested in either the development or performance of cosmetic surgery.

The question is often asked, what is the ethical difference between doing an abdominal operation and removing wrinkles from a sagging face? The answer is simple: The abdominal operation is necessary to the health of the patient; the operation for removal of wrinkles is unessential and is simply decorative surgery. So these conditions cannot be exactly compared.

However, there is a group of patients who desire face-lifting

[1] Cosmetic (definition from the *Century Dictionary*): Pertaining to beauty; beautifying; improving beauty, particularly the beauty of the complexion. . . Any preparation that renders the skin soft, pure and white, or helps or professes to be able to help to beautify or improve the complexion. . . . The art of anointing or decorating the human body, as with toilet preparations.

or some similar procedure done, who have a definite psychosis, and at times the plastic surgeon is called upon to operate in order to relieve the mental condition. These patients are seldom satisfied, however good the result may be, but nevertheless they have to be taken care of, as to them the unessential face operation means more than the relief of a severe abdominal condition.

Training. I feel very strongly that it is imperative for the surgeon who expects to do plastic and reconstructive work to have a thorough general surgical training, including a good working knowledge of anatomy and pathology, before attempting to specialize in this branch.

The plastic surgeon should be more than a skilful operator. He should know how to care for large unhealed defects and how to prepare them for contemplated plastic work, although it has been said by those who should know better, that the treatment of wounds is a waste of time for the plastic surgeon and is out of his field. He should understand the proper preparation of scar-infiltrated tissues and the utilization of such tissues. Thorough familiarity with the free transplantation of skin, mucous membrane, fat, fascia, bone and cartilage is essential, as all of these tissues are constantly utilized in reconstructive work. The principles of tissue-shifting and of the use of pedunculated flaps must be understood; also the possibilities of combinations with the above-mentioned free transplants.

The plastic surgeon has a point of view which is essentially different from, say, that of the man doing general surgery, as he has to deal almost entirely with defects and deformities of one sort or another, and in many instances his work is with tissues poorly nourished and infiltrated with scar. His operative methods also differ materially in many ways from those in common use in general surgery. He is accustomed to undertake cases which seem entirely beyond help and which have been refused or botched by others.

It is essential that he have an aptitude for the work. He should have sound surgical judgment and be a good operator. He should understand the use of local anesthesia. He should handle tissues with great consideration. The ability to study out and diagnose the condition in each case is necessary, as there are seldom two alike. An artistic sense is useful. He should know how to make haste slowly and not be tempted to do too much at a time or to do

it before the tissues are ready. He should understand the process of gradual building up, the entire series of operations being planned with regard to the ultimate result rather than the immediate relief of the condition. He should also know when to act swiftly. He should be skilled in postoperative care, as the success or failure of many plastic procedures depends on proper treatment after the patient is back in the ward. He should understand the surgical handling of children. He should have a knowledge of the use of prosthetic appliances and be familiar with the ordinary surgery of the nose, throat and mouth. In addition to his other qualifications, he must have originality and infinite patience and must understand the psychology of patients under treatment over long periods of time. He must be an optimist, something of an idealist and must have a far vision. Finally, he should have cheerfulness and a full measure of sympathy, of which he must give freely.

Diagnosis. Inasmuch as many of the lesions dealt with in plastic surgery can be seen, at least in part, the type of diagnostic skill required by the plastic surgeon is not the same as that which is necessary, for example, to determine obscure abdominal or brain lesions. Nevertheless, accurate diagnosis is of equal importance in plastic surgery, as perfect understanding of the extent of destruction or change in form in the structures involved is essential as well as the realization of the physical and mental effect of the lesion. In many instances this is far less simple to determine than it would seem to one unfamiliar with the difficulties involved and requires very circumspect handling, much thought and the aid of models, roentgen rays and many of the routine diagnostic methods. The keenest surgical judgment is often necessary to determine what should be done and how to do it; whether or not a plastic procedure should be finished at one operation; how far to go in the initial operation, and when to follow with the secondary procedures.

Results. The results obtained in many of the deformities corrected are little short of marvelous; but where there has been extensive destruction of tissue with much surrounding scar infiltration, it is rather far-fetched to claim that the part can be made to look exactly like it was before the accident. However, the improvement obtained in the majority of cases is well worth striving for.

RESEARCH. The field of research in plastic problems is a large one, and while very little stress has been laid on this aspect of the matter, considerable progress has already been made, and we are endeavoring to advance the knowledge of the subject along rational experimental lines. Much of this work should be done in the experimental laboratories, but there are important research problems to be worked out in the operating rooms and wards, which also require the trained investigator. For example, I might mention that there is still much to be learned about the utilization of transplanted tissues and their fate; the processes of repair; the chemistry of healing; the practical use of tissue cultures; the causes of congenital malformations, and so on *ad infinitum.*

The literature of plastic surgery is increasing very rapidly and numerous articles are appearing in many journals. Might it not be worth while to consider the publication of a special journal devoted to plastic surgery in its various phases similar to those of other surgical subdivisions?

What is the financial return which may be expected by a well-trained surgeon who limits his work to general plastic surgery, is a question which is often asked by young men who are contemplating taking up this branch of surgery? Not as much, to be sure, as is made by a very popular general surgeon, but enough for all practical purposes. It is impossible for any patient to pay in dollars and cents for the services rendered in some of the long-drawn-out cases, but I can say that there is a personal satisfaction in the successful outcome of this work which I do not think any other division of surgery gives. I believe that service to others is the watchword in legitimate plastic surgery even more than in other branches of surgery. It is true that the deformities dealt with by the plastic surgeon are found more frequently among those not blessed with a superabundance of this world's goods, but if these patients can be put back to making their living and can go about without attracting undue attention on account of their disfigurement, then something really worth while has been accomplished.

If one eliminates the ideals of ethical plastic surgery, and goes into what is known as cosmetic or decorative surgery, then the financial picture changes. The incomes made by some of these men, who are mostly quacks, are said to be enormous, as the individuals who want face-lifting and similar operations done usually have money to pay for it, and as a matter of fact, I am told, have to pay for it in advance or it is not done.

TEACHING. The teaching of plastic surgery is essentially post-graduate. The medical student should be given enough to familiarize him with the scope of the subject and he should know in a general way what can be done in this field. In this branch, as in all others, the real training only begins after graduation, and so, with the hearty coöperation of Dr. Lewis, we are beginning this fall to combine a general surgical training with special attention to general plastic surgery. An assistant resident who is interested will be assigned to the work and will be carried through until proficient. This plan seems the best so far developed. We are fortunate at the Johns Hopkins Medical School in having two fellowships in plastic surgery granted by the General Education Board, which are assigned to carefully picked graduates. Their research training is carried on in the Hunterian Laboratory and their clinical experience is obtained in the plastic surgery dispensary, in the wards and operating rooms. The great need with us at present is ward beds, but the prospects for the future are promising, and we feel that in a comparatively short time this lack will be supplied. Elsewhere, as far as I can ascertain, there has been as yet no systematic attempt to develop the teaching of general plastic surgery.

PRESENT STATUS OF PLASTIC SURGERY. The present status of those doing plastic surgery in the United States and also in Europe is about as follows: A few well-trained surgeons, who have given special attention to plastic surgery of the entire body, who can be classed as general plastic surgeons; those general surgeons, who have a special talent for the work, but ordinarily have not the time or inclination to give to it; a larger group, war trained, who specialize in plastic surgery of the face, and some of them are doing splendid work; then a mixed group, who confine their plastic surgery to one type of operative work, as, for instance, the nose, the eyelids, congenital clefts of the lip and palate. Then the group of surgeons without special talent or knowledge who do plastic operations as they do everything else that comes under their hand. Those who specialize in cosmetic or decorative surgery should not, in my opinion, be considered plastic surgeons.

AS A CAREER. The question of taking up plastic surgery as a career is a very different matter today from what it was twenty years ago, when I essayed to do it. To begin with, the opposition to its full development is now small; it is an established branch;

there are a number of excellent books available; there are better facilities for seeing good operative plastic work; there is a great popular and professional interest in the subject. It is possible to get instruction; there are certain fellowships available to suitable men desiring to perfect themselves. There is an association of plastic and oral surgeons. The nomenclature is being simplified and standardized. None of these advantages were at hand twenty years ago.

After this brief outline of the subject the question arises, is the field of plastic surgery large enough and interesting enough to justify a trained surgeon in devoting his entire time to it?

I am convinced that it is. As the years go on I find that the field seems to grow wider and the work becomes more useful in the scheme of a general surgical department with its various subdivisions.

Future Development. I believe that very soon there will be created in every large surgical clinic a subdivision of plastic and reconstructive surgery, such as we now have at the Johns Hopkins Hospital, under men especially fitted and trained for this work.

I am now confidently looking forward to the time when this division can send out men adequately trained in general plastic surgery from the standpoint of the laboratory, the wards and the operating rooms, and to this I hope to be able to add some common sense and a good deal of the milk of human kindness.

THE CAUSES OF CICATRICIAL CONTRACTION

By J. SHELTON HORSLEY, M.D.
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Not infrequently there has appeared in surgical literature a statement, or at least a suggestion, that defects in the common bile duct or in the ureter would be satisfactorily repaired simply by epithelium covering the defect. This idea has been the basis of procedures in which a rubber tube or a catheter was placed in the common duct or in the ureter, expecting the epithelium to grow over the tube from each end of the divided duct or ureter and that when the epithelial covering was complete there would be practically perfect repair. Or tubes of fascia or a segment of vein would be used with the expectation that after the epithelium had covered the interior of the transplant, permanent function would be established.

Strictures, however, are usually covered with epithelium. The histologic examination of a stricture, or of any cicatricial contraction about the face, often shows an epithelial covering that is not far from normal. Hunner and Wharton have sections of blocks from a stricture of the ureter in which the epithelial coat is preserved and the pathologic changes are in the other tissues. In cicatricial contractions about the head and neck, which are common after burns, the epithelial covering is practically normal but the contraction is due to the scar tissue beneath. The maximum contraction usually occurs after the surface has healed and is entirely covered with epidermis.

A little white girl, F. K., aged five years, admitted to the hospital December 16, 1925, was burned one year before admission. There was on the left side a very severe contraction of the head and neck, and the upper arm was adherent to the body (Fig. 1). A plastic operation was done and the excised scar tissue was studied histologically. Even in the region of greatest contraction and densest scar tissue the epidermis was practically normal.

A comparison of sections from an area of this contraction with those from normal skin is interesting. Fig. 2 is a photomicrograph of normal skin from the chest of a woman operated upon for mammary carcinoma, the skin being taken from the margin of the gross specimen. When compared with Fig. 4 from the densest mass of scar tissue in the neck (Fig. 1), but little difference can be seen in the two epithelial coverings. The contraction is due

FIG. 1.—Frances, K. Deformity from cicatricial contraction of the left side of neck and the left pectoral region.

to the replacement of the true corium by dense scar tissue. As corium consists chiefly of connective tissue and the scar is connective tissue, the histologic difference between these structures is not marked. Fig. 6 and 8 show sections from the pectoral and the axillary contractions of F. K. (Fig. 1). There is an essentially normal epithelial covering. Sections from cicatricial contractions elsewhere in this patient present the same type of epidermis.

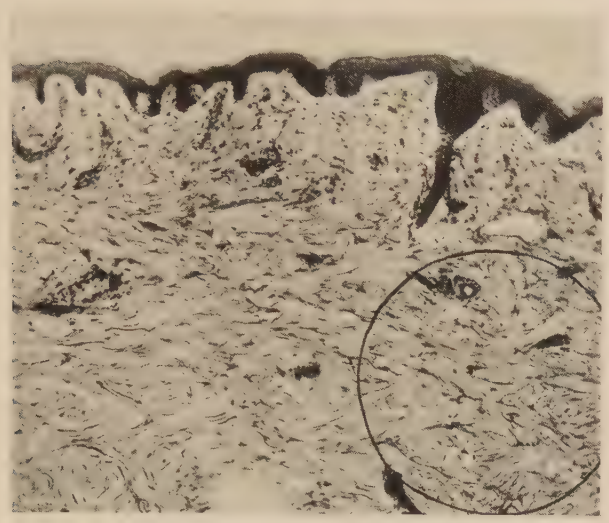

FIG. 2.—Mrs. F. Normal skin from chest. Note the epidermis, with a section of a duct to a sweat gland which has been cut partly obliquely, and the structure of the connective tissue of the corium. (× 60.)

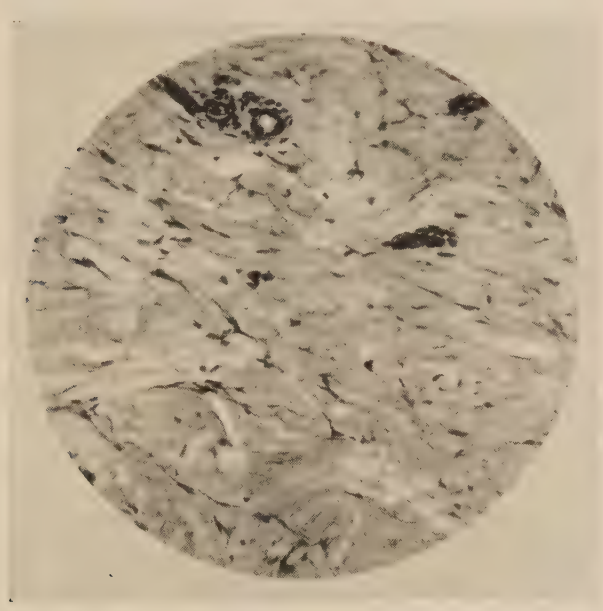

FIG. 3.—Mrs. F. Higher magnification of the connective tissue in the preceding photomicrograph. (× 150.)

Figs. 11 and 12 are from a section of a scirrhous cancer of the breast in which there was marked contraction. The connective tissue is in very dense bands; there are occasional areas of apparently degenerated tissue and some carcinomatous cells arranged in atypical acini. Fig. 13 is a photomicrograph of a section from a cirrhosis of the liver in which there was contraction. The con-

Fig. 4.—F. K. Photomicrograph of the dense cicatricial tissue from the neck of patient shown in Fig. 1. Note the comparatively normal epidermis. The connective tissue shows regions of leukocytic infiltration. (× 60.)

nective tissue is in approximately straight fibers with considerable leukocytic infiltration due, doubtless, to the irritating effect of the toxic substance that caused the cirrhosis. In a slowly growing carcinoma of the colon, in which there was marked cicatricial contraction, the photomicrograph shows connective tissue much of which is wavy (Fig. 14).

When the histologic structure of a contracting scar (Figs. 4, 5, 6, 7, 8, 9, 10, 11, 12, 13 and 14) is compared with a scar in which there is no contraction (Figs. 15, 16, 17 and 18), it is seen that there is no essential difference. The epidermis over a noncontracting scar is also apparently normal. In Fig. 18, scar tissue of a noncontracting scar is in contact with the normal corium. The tissue is from the abdominal scar of an operation done two years ago, the scar being excised during a later operation. The tissue in a noncontracting scar in the breast (Fig. 15) contains

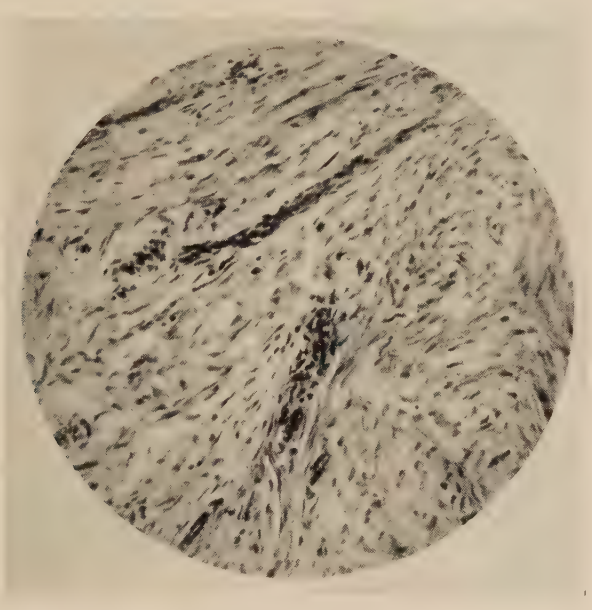

FIG. 5.—F. K. Higher-power view of another field of the same slide from which the preceding photomicrograph was made. (× 150.)

some of the wavy type of connective tissue. Theoretically it would seem that the wavy type in which there is contraction of the total length of the connective tissue fibers would produce the greatest contraction. This type was found in carcinoma of the bowel (Fig. 14) and in some of the sections from cicatricial contraction of the neck (Figs. 6 and 7), but it does not show in the scirrhous cancer of the breast (Figs. 11 and 12) nor in the cirrhosis of the liver (Fig. 13). It also occurs in the noncontracting scars (Figs. 15, 16, 17 and 18).

Various types of connective tissue are found in cicatricial contraction. In Figs. 6, 7 and 14 there is a wavy type, but in Figs. 5, 8, 10, 11 and 12, also from an area of contraction, the connective tissue is in dense straight bundles. When compared with sections of normal skin (Figs. 2 and 3), or of noncontracting scars (Figs.

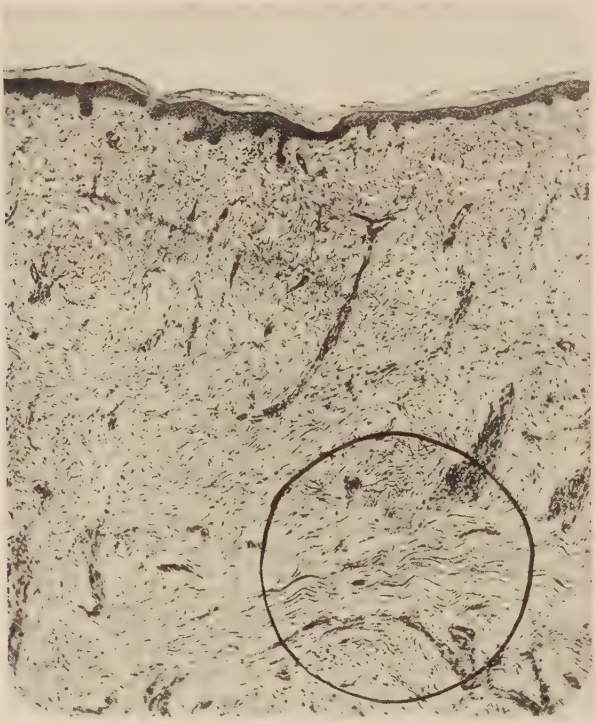

Fig. 6.—F. K. A section from the upper pectoral region of the contracting scar (Fig. 1) showing the epidermis practically normal, with connective tissue arranged in dense columns in the upper portion of the photomicrograph and in wavy bundles in the lower portion. (× 60.)

15, 16, 17 and 18), no fundamental histologic difference in the connective tissue can be seen.

A histologic comparison of the connective tissue from all these sections does not seem to give any clew to the cause of contraction. It appears that we must look elsewhere than to the microscopic study of the connective tissue constituting the scar.

The origin of connective tissue has been the subject of interesting research in recent years. George A. Baitsell seems to have shown that connective tissue in the chick embryo and in the amphibian begins as a "transparent, gelatinous, cell-free ground-substance which, in general, pervades the embryonic body from very early stages of development," and which he thinks is a secretion of the cells of the various germ layers and has no connection with a syncytium or any transformation of the cytoplasm.

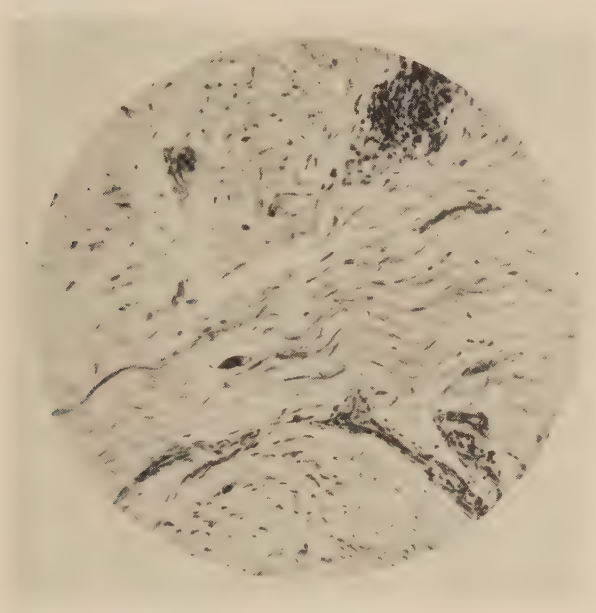

FIG. 7.—F. K. The connective tissue from the lower portion of the preceding figure, showing the wavy fibers of the connective tissue with regions of leukocyte infiltration. (× 150.)

Fibrillation later occurs in this ground-substance and increases as the embryo grows. "The formation of the ground-substance is followed by the invasion of the mesenchyme cells which, using it as a supporting material, apparently in the same way that cells utilize the plasma clot in tissue culture, move through and modify it in various ways."

Hertzler has found that in certain conditions peritoneal healing may occur by direct transformation of the fibrin without the intervention of granulation tissue. Where there is a distinct granula-

tion tissue, however, the process of healing is more complicated and there seems to be a transformation of the cells of the granulation tissue into connective tissue, though some of the connective tissue may be from direct changes in the fibrin, as has been suggested by Hertzler and by Baitsell. According to H. E. Jordan,

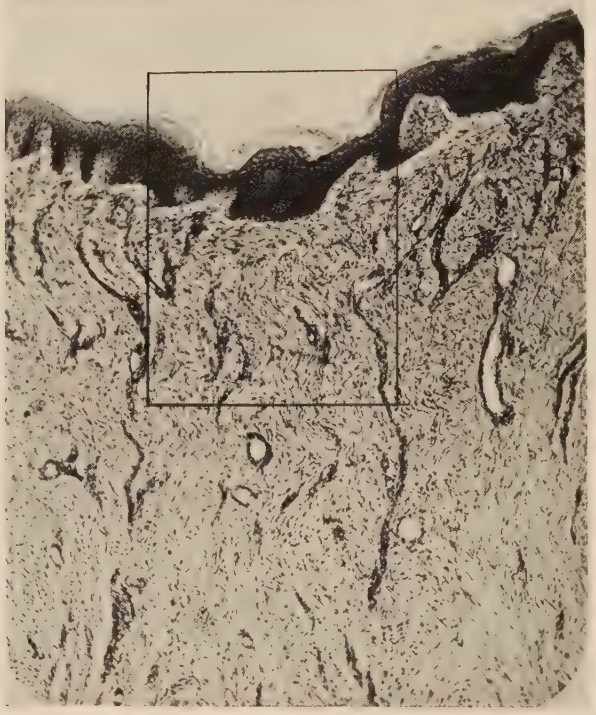

FIG. 8.—F. K. A section from the axillary region of the contracting scar tissue (Fig. 1), showing normal epidermis, with connective tissue in wavy formation arranged at right angles to the epidermis, with many newly formed vessels. (× 60.)

of the University of Virginia, lymphocytes are the chief elements in the organization of scar tissue. Carrel has shown that leukocytes have a growth-promoting function. Jordan and Speidel have shown that this "trephonic" action is probably located in the lymphocytes. Moreover, Carrel and Ebeling have demonstrated by tissue culture methods that lymphocytes may differen-

tiate into fibroblasts, confirming the earlier claims of Policard and Desplas and of Renaut.

According to H. E. Jordan, the fibroblasts of the developing scar tissue apparently have origin chiefly from lymphocytes, in small part from the local connective tissue cells stimulated to

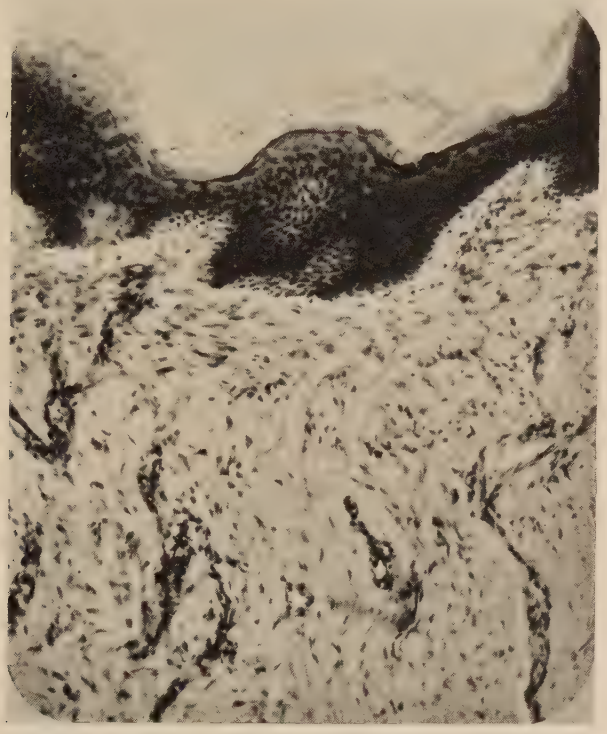

FIG. 9.—F. K. Higher-power view of the epidermis and scar tissue immediately beneath it, shown in the preceding photomicrograph. The scar tissue is dense and immediately beneath the epidermis is parallel to the surface, but beneath this layer columns appear at right-angles. (× 150.)

proliferative activity by the presence of leukocytes, and possibly in very small part also from endothelium (Mallory). The variable origin of the fibroblasts from lymphocytes, connective-tissue cells and endothelial cells is explicable on the basis of the close genetic relationship among these only slightly differentiated elements of the original mesenchyme.

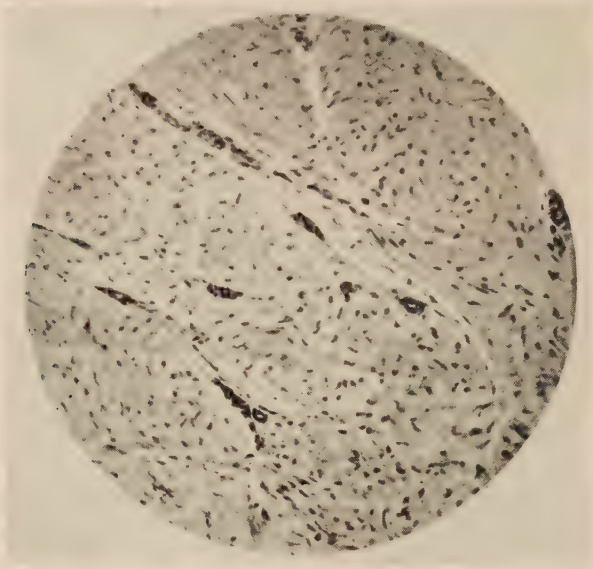

FIG. 10.—F. K. Connective tissue from the posterior axillary region of the contracting scar (Fig. 1). The bundles, many of them in cross-section, seem dense, with no tendency toward wavy formation. (× 150.)

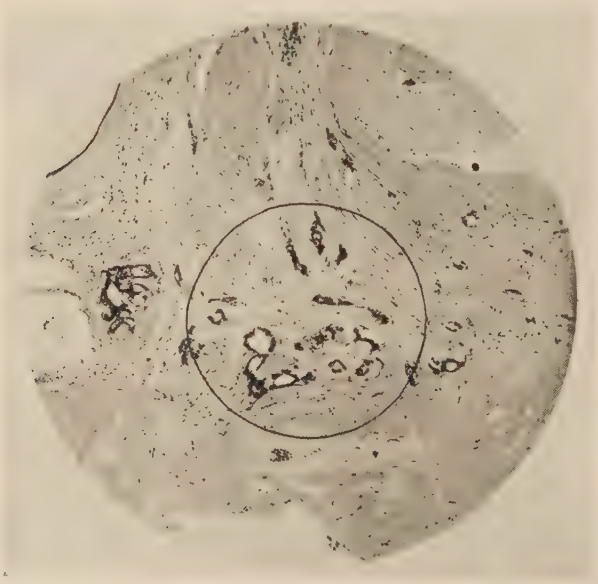

FIG. 11.—Mrs. M. A section from a scirrhus of the breast. There are dense connective-tissue bundles, with their fibers straight, areas of degenerated tissue and atypical acini of cancer cells. (× 60.)

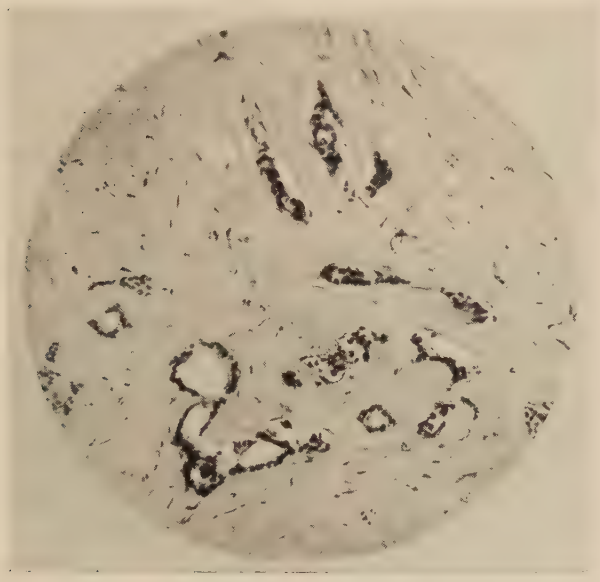

FIG. 12.—Mrs. M. Photomicrograph giving higher-power view of a portion of Fig 11. The connective tissue shows well in the upper right-hand side, and is exceedingly dense. (× 150.)

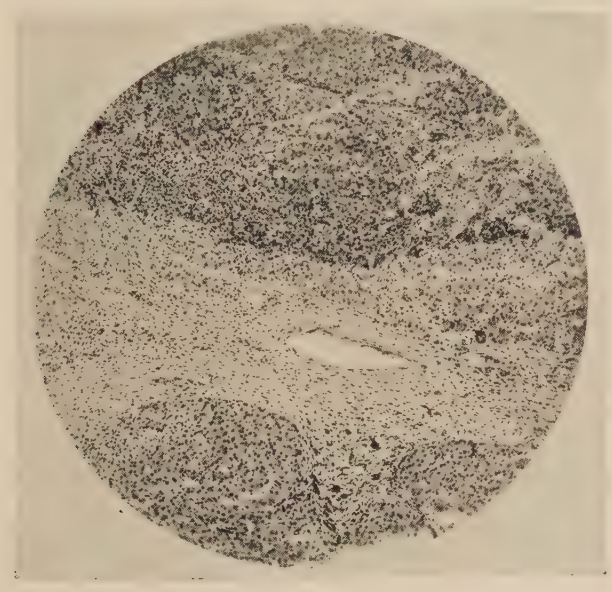

FIG. 13.—A section from cirrhosis of the liver. The connective tissue is rather dense, with straight fibers. There is considerable leukocytic infiltration. (× 60.)

Scar tissue is a type of connective tissue which is a low order of tissue and may be compared with more highly differentiated tissues, as the invertebrates in a zoölogical classification are compared with mammals. The lower order of animals shows a high capacity for repair and the ability to live under conditions that would be fatal to the more complex higher animals. The different

FIG. 14.—Mrs. S. A slowly growing cancer of the colon which produced a marked stricture. Note the wavy lines of the connective tissue, which is exceedingly dense. (× 150.)

tissues in man also have varying capacities for surviving injuries and for repair. The delicately constructed and more recently evolved cortical brain-tissue repairs not at all, whereas connective tissue, one of the primal tissues, repairs almost perfectly. Between these two extremes there are different degrees of ability to repair. When conditions are such either from toxic or traumatic injury

or from lack of nutrition that more highly differentiated tissues cannot survive, connective tissue often may live.

While there is no direct relation between an epithelial covering and cicatricial contraction, because contraction is due to the connective-tissue element, the epithelial covering may have an indirect effect. If a granulating surface persists for a long time, there is a tendency for an excessive amount of connective tissue to form. The soft granulation tissue is easily traumatized and

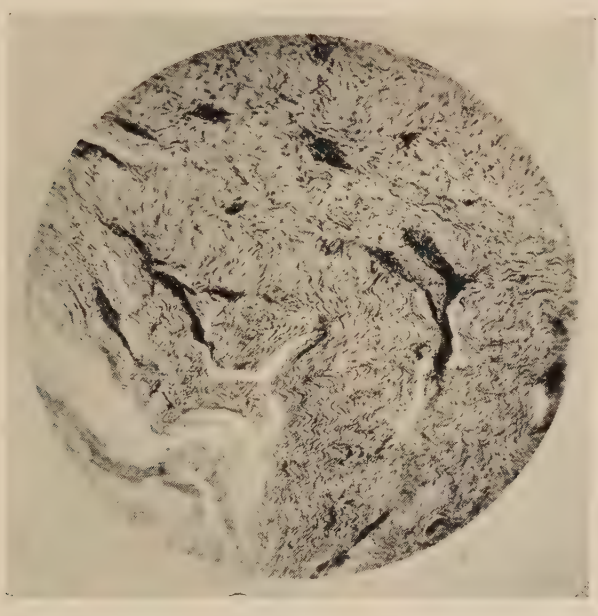

FIG. 15.—Miss E. R. Connective tissue from a scar on the breast. The scar was moderately broad, covered with normal epidermis, but was not contracted. (× 60.)

each trauma is a stimulus for additional granulation tissue and subsequent connective-tissue formation. If this raw surface is covered with epithelium, as with Thiersch grafts, the epithelium acts as a protection from further trauma. After scar tissue has formed, however, the epithelial covering does not prevent contraction. If cicatricial contraction in the skin is to be corrected the whole skin should be used as a transplant. Where the normal corium is thin and the nutrition good, as in the eyelids, there may

form under the Thiersch grafts a new corium of connective tissue which will have but little tendency to contract. Thiersch grafts do not, as a rule, prevent a recurrent contraction, though they may in some instances mitigate it.

The causes of cicatricial contraction can be classified roughly under two heads, direct and indirect. The direct cause is a toxic substance which in the last analysis is, of course, chemical and

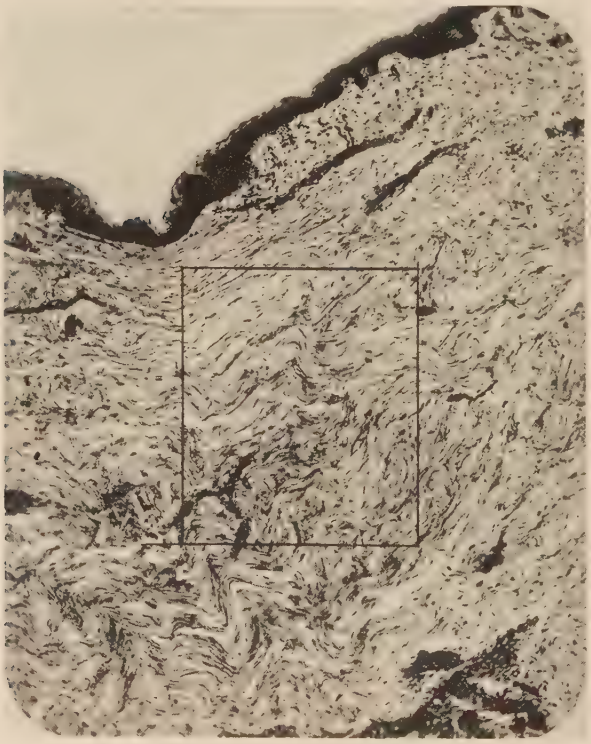

FIG. 16.—Mrs. W. E. G. Noncontracting scar from the abdomen. Note the apparently normal epidermis and the wavy connective tissue. (× 60.)

consists of products formed outside of the body cells, as from bacteria, or produced within the cells, as when they are affected by radiant energy. The indirect causes may be considered as predisposing.

1. *Direct Causes of Cicatricial Contraction: Toxic Products Produced by,*

(*a*) Burns (by heat, light, or electricity).
(*b*) Chemicals.
(*c*) Bacteria.
(*d*) Cancer.
(*e*) Trauma and cells affected by lack of blood supply.
(*f*) *X*-ray or radium.

2. *Indirect Causes:*

(*a*) The general disposition of the individual toward scar-tissue formation.
(*b*) The portion of the body affected.
(*c*) The absence of strain or tension on the scar.
(*d*) Lack of proper blood supply.
(*e*) The absence of natural resistance toward physiologic secretions or excretions.
(*f*) The quantity of scar tissue.

1. (*a*) Cicatricial contraction is particularly noticeable after burns. This is probably chiefly due to the fact that along the margin of the completely destroyed tissue there is a zone in which the cells are injured but not killed, and sooner or later the more highly differentiated of these cells will be replaced by connective tissue. The toxic product from burns may result partly from destruction of the cells and partly from substances developed within those cells injured but not completely destroyed. Often a burn is complicated in healing by infection when the toxins of bacteria are added.

1. (*b*) Toxic products from chemicals, as after injuries from strong acids or alkalies, doubtless are formed in much the same way as from burns, the chemical itself having a directly destructive action with a zone in which cells are injured but not completely destroyed.

1. (*c*) The toxic products from bacteria are probably chiefly elaborated from the bacteria themselves, but partly also from contact of these toxins with living tissue. The effects of bacterial toxins are often rather far-reaching. Aside from infection of a wound, which causes necrosis of the local tissues and injury of the adjoining cells, milder toxic bacterial products gain access to the circulation and sometimes seem to have a specific action upon certain tissues. Thus, cirrhosis of the liver (Fig. 13) may be caused

by the toxins of the colon bacillus or from toxic products supplied through the spleen as well as from the chemical toxic products from alcohol or chloroform. Dupuytren's contraction, due to cicatricial bands in the palm of the hand over the flexor tendons of the little and ring fingers formed without apparent trauma, has been puzzling. Leonard W. Ely, of Stanford University, thinks the first step in the treatment of Dupuytren's contraction

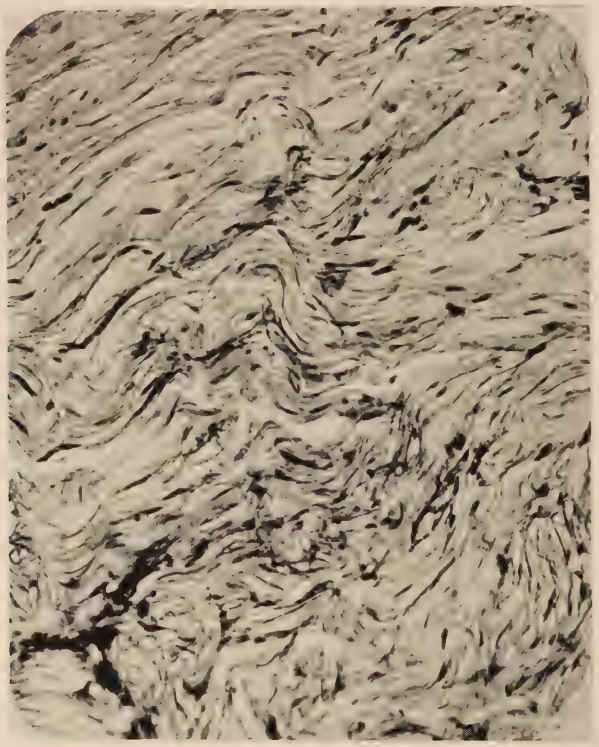

FIG. 17.—Mrs. W. E. G. Higher-power view of the connective tissue shown in Fig. 16. The fibers of the connective tissue are fine, lace-like but rather dense. (× 150.)

should be the removal of foci of infection in the teeth or in other portions of the body with the idea that bacterial products from these foci have an affinity for the fascia of the palm of the hand. The affinity of certain streptococci for the mucosa of the pyloric portion of the stomach and for the duodenum has often been

emphasized by Rosenow, who believes these bacteria cause peptic ulcers and inflammation in these regions—lesions which not infrequently result in cicatricial contraction and stenosis.

1. (*d*) Malignant disease causes reaction in the surrounding tissue. If a cancer grows rapidly, cells and nutrition are abundant and the disease may run its course before there is opportunity for

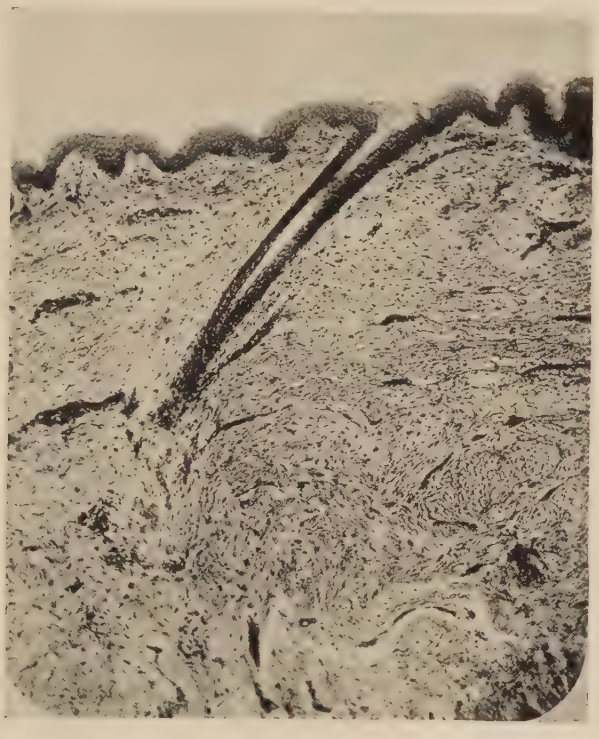

FIG. 18.—Mrs. W. E. G. Noncontracting scar of the abdomen. The union of the scar on the left with normal skin on the right is shown. The epidermis over the scar appears identical with that over the normal skin. Near the middle of the photomicrograph is a hair follicle. (× 60.)

the secondary and slower changes of mature connective-tissue formation to occur. In the more slowly growing cancers, however, the reaction from the toxins elaborated during the growth of the cancer cells causes marked connective-tissue formation and cicatricial contraction within the tumor itself. This is commonly

seen in the breast, as scirrhous cancer, and in certain cancers of the large bowel (Figs. 11, 12 and 14).

1. (*e*) Trauma may produce a defect by removing or destroying a large mass of tissue, or may injure only a few cells. Between these extremes there are many degrees of injury. Trauma seems

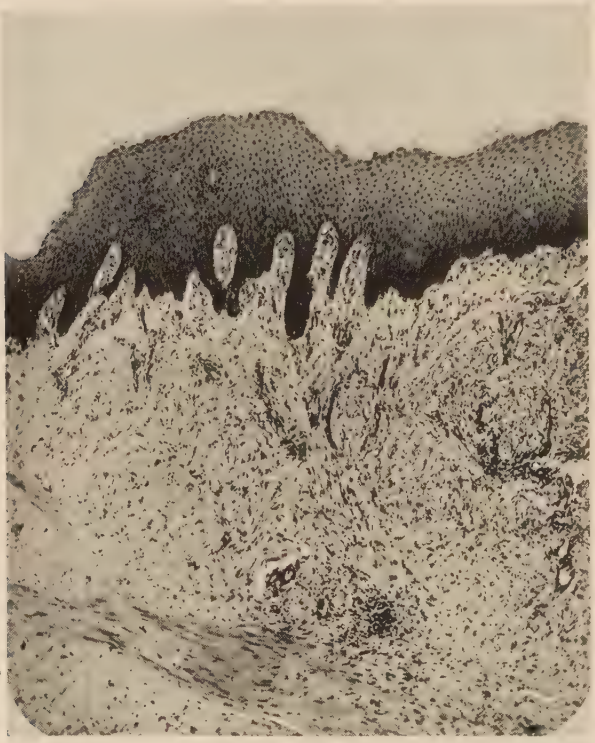

FIG. 19.—Dr. W. H. S. Photomicrograph of scar resulting from *x*-ray burn. The epidermis is thicker and tends to grow down into the tissue more than in the other scars shown, which resulted from a burn or trauma. The scar tissue beneath the epidermis is moderately dense, with considerable leukocytic infiltration. (× 60.)

to produce toxins by its effect on the tissue cells which in their degeneration release these products. If this were not true the stimulus for repair would not occur. Multiple and frequently repeated mild traumas before scar tissue has fully developed tend to increase scar-tissue formation; but after the maturity of

the scar a mild repeated trauma, such as massage, often causes some absorption of the scar tissue. Tissue cells necrotic or degenerating from lack of blood supply, doubtless produce toxic products as do cells affected by trauma.

1. (*f*) Cicatricial contraction following *x*-ray or radium burns presents several interesting problems. These scars are usually very painful. The pain apparently is largely ischemic and due to the lack of blood supply, which in turn seems to be induced by

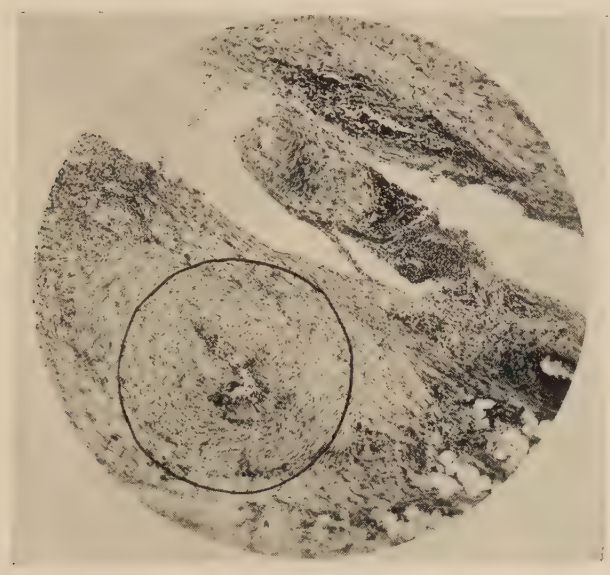

FIG. 20.—Dr. W. H. S. Photomicrograph of a bloodvessel from the scar tissue just beneath that shown in Fig. 19. Through the upper portion of the tissue there is an artifact caused by the section pulling apart when it was cut. (× 60.)

the action of the *x*-ray or radium in stimulating the endothelial lining of the bloodvessels and causing the endothelium to proliferate and occlude the vessels. As has been discussed before, scar tissue being able to survive when more highly differentiated cells cannot, the resulting lesion from intensive application of *x*-ray or radium, which injures tissue cells and so produces toxic products, consists largely of scar tissue. In Fig. 19 is shown a slightly contracting scar following an *x*-ray burn. In this patient, a

dentist who did his own *x*-ray work, the inner side of the thigh had been frequently exposed to the *x*-ray. There followed ulceration which healed except at the central portion. An examination of the excised tissue showed at one small point squamous cell cancer. In other areas there was merely hypertrophy of the epithelium, beneath which was scar tissue with obliterated vessels. Figs. 20 and 21 show one of the vessels almost entirely obliterated apparently by proliferation of its intima. In scar tissue from burns

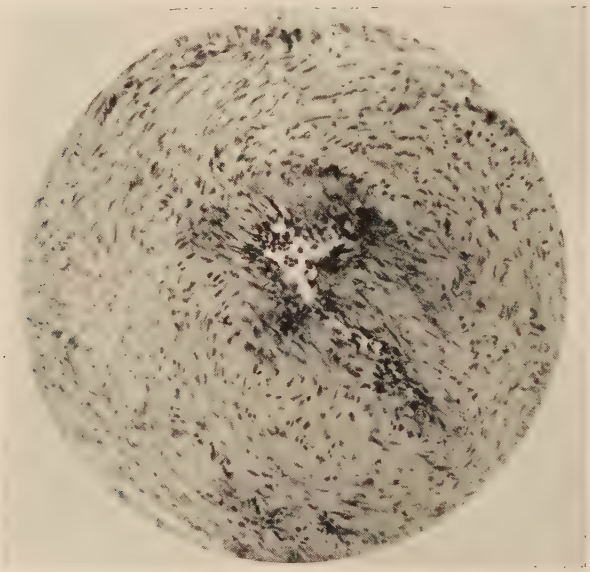

FIG. 21.—Dr. W. H. S. Higher-power view of the bloodvessel shown in Fig. 20. The lumen is almost occluded, apparently by growth of the intima. There are faint outlines of what seems to be the inner elastic membrane. (× 150.)

or bacterial infection, there appears to be no unusual effect upon the endothelial lining of the bloodvessels. It is well known, too, that scars from *x*-ray or radium lesions are peculiarly prone to develop cancer. Montrose T. Burrows, of St. Louis, has elaborated a theory that cancer is initiated by an impaired circulation which permits stagnation around a group of cells of a by-product of the cells called archusia, corresponding to vitamin B. It is interesting to note that in the only type of scar in which the circulation is

peculiarly impaired by occlusion of the vessels there is a decided tendency toward cancer. Without necessarily endorsing this theory of Burrows, the coincidence of these conditions demands attention.

FIG. 22.—Section from portion of a vein that was transplanted to fill a defect of the common bile duct of a dog. The vein was everted so that the internal surface of the transplant of vein consisted of the adventitia and lined the lumen of the transplant. This is shown on the right, whereas the external surface of the transplant on the left consists of intima. Note the marked infiltration of the internal lining of the transplant on the right. (× 30.) (From Jour. Am. Med. Assn., 1918, lxxi, 1188.)

2. (*a*) Among the indirect causes of cicatricial contraction is a certain idiosyncrasy. In some persons scar tissue forms more readily than in others. A small scar, for instance, which is merely linear soon after an operation, weeks or months later without

apparent cause may develop into a large, broad, scar. This is not always accompanied by contraction, and frequently such a scar is quite cellular, and is called a keloid. In some true keloids, however, even when there is no contraction, the connective tissue appears very dense.

2. (*b*) The portion of the body involved has much to do with the development of cicatricial contraction. It is well known that scars in certain regions of the body do not contract, whereas in other regions scars of apparently similar nature produce marked contraction. Every surgeon avoids a longitudinal scar in the

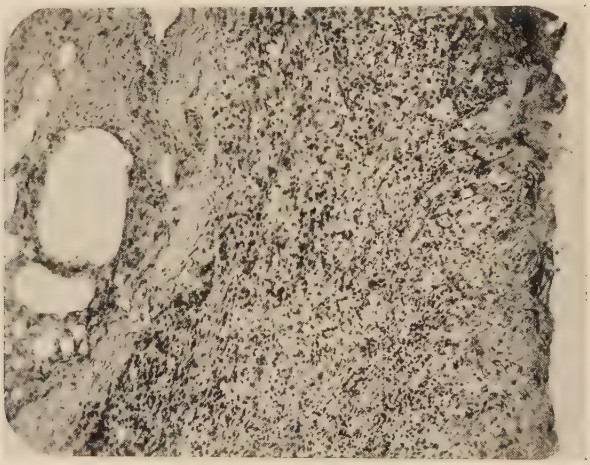

FIG. 23.—Higher-power view of internal surface of transplanted vein shown in Fig. 22. Note the marked infiltration of the adventitia with small round cells. (× 125.) (From Jour. Am. Med. Assn., 1918, lxxi, 1188.)

palm of the hand, in the axilla, or in the middle of the neck, whereas a transverse scar in these regions does not contract. Longitudinal scars on the flexor surfaces as in the popliteal region, in the front of the elbow, and particularly in the axilla or on the anterior surface of the neck, are very prone to contract. Longitudinal scars in the axilla or in the anterior surface of the neck seem to be more likely to contract than in the popliteal region or in front of the elbow. This may be due to the fact that in these latter regions the normal position for the limb at rest is fully extended, whereas in the front of the neck or in the axilla the normal resting posi-

tion is for the head or the arm to be flexed; they are never fully extended at rest.

2. (*c*) An indirect cause of cicatricial contraction is the absence of pressure or tension. This is a noted feature of scar tissue, that it will tend to contract when there is no strain on it, or to stretch when there is. In this respect it differs somewhat from normal connective tissue with which it may be histologically almost identical. Though frequent traumas in the process of

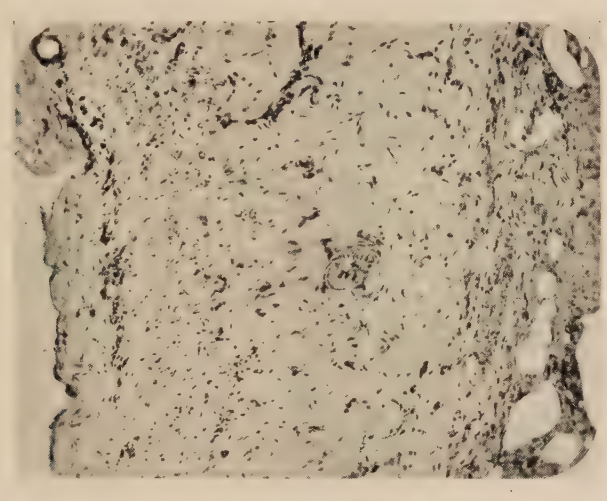

FIG. 24.—Photomicrograph of outer wall of transplanted vein shown in Fig. 22, corresponding with the everted intima and a portion of the media of the vein. This represents the tissue on the left side of Fig. 22. This tissue seems well preserved and contains but little inflammatory products when compared with the portion of the transplanted vein which was bathed in bile. (× 125.) (From Jour. Am. Med. Assn., 1918, lxxi, 1188.)

healing may add to the scar, later when the scar is mature, measures that produce increased circulation and increased metabolism not infrequently cause some of the scar tissue to disappear. The tension on an abdominal scar may induce such a change, and so weaken the whole scar. In certain locations intermittent stretching tends to decrease the scar and to make it give way. Strictures of the urethra, for example, are often cured by the occasional passage of a sound and the cicatricial tissue, from being rather dense at the stricture, may almost entirely disappear. The tension

on the scar of an abdominal wound, however, is almost constant. Before the Bassini operation came into vogue the late Charles McBurney devised an operation for the cure of hernia which was founded on excessive scar-tissue formation. Dr. William B. Coley in a letter of May 6, 1926, has given me his personal recollections

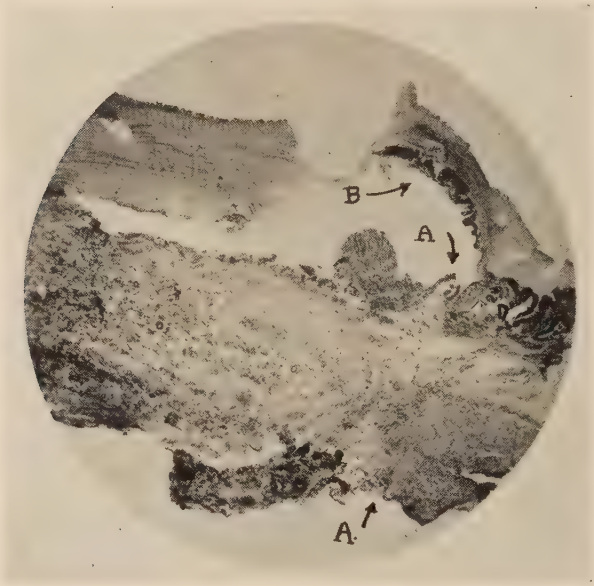

FIG. 25.—Photomicrograph of the junction of a transplanted vein at the stump of the common duct (*A*, *A*). In these experiments a segment of the everted vein was used to reconstruct the defect in the common bile duct of a dog. This specimen was removed twenty-three days after operation. The normal duct tissue is on the right and the transplant is on the left. The junction is shown by arrows (*A*, *A*). The columnar epithelium at *B* has partly grown over the transplant, but its attachment was slight and was partly broken while making the section. (× 30.) (From Jour. Am. Med. Assn., 1918, lxxi, 1188.)

of this operation, as he was house-surgeon in the New York Hospital at the time McBurney brought it out. The technic consisted in leaving the wound widely open and stitching the skin on either side to the bottom of the wound so that granulations came from the bottom up. The entire wound was then carefully packed. The operation was based upon the idea that the dense scar tissue

would form a barrier against recurrence. Dr. Coley says: "As a matter of fact, however, the scar tissue very quickly began to yield and in most of the cases in which the operation was performed at the Ruptured and Crippled, a recurrence took place within the next few years."

2. (*d*) The lack of proper blood supply may predispose to the formation of cicatricial contraction. This sometimes acts by lowering the resistance of the tissues to bacterial invasion and so increasing the scar, or, in a transplanted flap, even without infection, the nutrition may be at so low an ebb that while the more highly differentiated tissue cannot survive, the lowly scar tissue will flourish and dominate. A striking illustration of the replacement of a higher class of tissue by contracting scar tissue is in atrophy of the testicle when its nutrition is impaired. This condition has been occasionally observed after extensive operations for varicocele when the blood supply to the testicle has been unduly impaired, and after operations for undescended testicle in which the spermatic artery has been purposely severed in order to relieve tension and permit the testicle to be brought into the scrotum. These operations have been followed first by swelling and later by atrophy of the testicle. Hermann B. Gessner, of New Orleans, in experiments in which he severed the spermatic artery in young dogs, usually found the testicle swollen a few days after the operation and later atrophied. There was no gangrene, but an eventual decrease of the epithelial parenchyma of the testicle occurred with an increase of connective tissue.

2. (*e*) Another interesting predisposing cause of cicatricial contraction is the absence of resistance of some tissues to certain normal secretions or excretions. This has been shown in attempts to repair the common bile duct. In a series of experiments in which I attempted to repair the common duct in a dog by transplanting a segment of everted vein, it was found that there was marked inflammatory infiltration of the transplanted vein with subsequent contraction and complete occlusion if the dog lived several months. This occurred regularly in the dogs that survived. Histologic examination of the transplant showed that the portion nearest the bile, the internal layer, was always deeply infiltrated with round cells while the outer coats were but slightly affected (Figs. 22, 23, 24 and 25). The lack of local resistance to a foreign environment is illustrated in the occurrence of jejunal ulcer after

gastro-enterostomy with an open pylorus. Mann and Williamson have reproduced in a dog a typical peptic ulcer by anastomosing the jejunum to the pyloric end of the stomach after diverting the alkaline duodenal contents. In many instances a peptic ulcer was formed where the mucosa of the jejunum was exposed to the gastric juice without the protection of the alkaline contents of the duodenum. The irritation of the skin of the abdomen by the discharge from a fecal fistula, especially a fistula high in the jejunum, shows the poor resistance of the normal skin of the abdomen to the normal jejunal contents, even though the skin may resist traumas that the soft velvety mucous lining of the jejunum could not withstand. In the reconstruction of any viscus we should always take into consideration this phenomenon of the adaptation of tissues to their environment. No matter how neatly the suturing is done, if the transplanted tissue has not some biologic relationship to the region in which it is placed and if it has no resistance to the secretions from the viscus to which it is transplanted, there will be marked reaction and consequently great scar-tissue formation and contraction. This is true not only of ordinary secretions, but to some extent of infection. Wounds in the perineum, for example, as from perineal prostatectomy, which must of necessity be exposed to infection from feces, usually heal primarily and without undue cicatricial contraction, whereas if a small fraction of the fecal matter which must occasionally contaminate peritoneal wounds be transplanted to a wound in the suprapubic region or elsewhere high up in the body, severe infection would result. It is not uncommon for bone in a fracture of the jaw bone, either complete from severe trauma or partial as from extraction of a tooth, to be exposed to the secretions of the mouth, and yet often but little if any infection occurs. In a compound fracture of the leg or arm, a similar exposure to the saliva and bacteria from the mouth would commonly result in sepsis.

2. (*f*) The quantity of scar tissue seems to have much to do with contraction. The greatest contraction is usually along the central mass of the scar (Fig. 1). For this reason it seems probable that lack of nutrition to the scar tissue causes some shrinkage of the cells which may tend to promote contraction. Occasionally there is found a narrow strong band of contraction. This is doubtless due to the survival from an original greater mass of scar tissue of the central portion which had become so well organized that

even when better nourishment caused absorption of some of the scar, the well-organized central portion remained.

SUMMARY. 1. Cicatricial contraction is due to scar tissue, and covering the surface of a wound with epithelium alone does not prevent contraction except so far as it prevents infection or unnecessary trauma to a raw surface.

2. The cicatricial contraction following *x*-ray or radium burns differs from other scars in that the deeply penetrating rays cause an overgrowth of vascular endothelium which partially or completely obliterates the bloodvessels and makes a greater degree of ischemia.

3. There is a biologic resistance possessed by different tissues of the body to the normal secretions or excretions of their environment, so that in transplanting tissue this must be taken into consideration.

4. Certain regions of the body, possibly because of some inherent quality or possibly because of mechanical conditions which retard complete extension, are more prone to scar-tissue contraction than other regions.

5. As scar tissue is a lowly tissue that can survive on less nutrition and under harder conditions than other more highly differentiated tissues, it seems essential in avoiding cicatricial contraction not only to prevent the irritating effects of frequent injuries or of toxic substances, but to provide a blood supply so that the higher tissues may survive and not be overwhelmed by scar tissue.

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SOME DETAILS IN THE REPAIR OF CICATRICIAL CONTRACTURES OF THE NECK

By CHARLES N. DOWD, M.D.
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The deformities which follow extensive burns and scalds in the neck are sometimes very distressing. These deformities have been frequently pictured in text-books and surgical journals. If, however, one looks for detailed descriptions of the successful surgical treatment of the condition, the search is disappointing. Not many such descriptions are to be found.

The following photographs and diagrams are published to illustrate certain details in the treatment of these contractures by various forms of skin flaps. This is done with full knowledge that some of my friends advocate full-thickness, detached grafts in these conditions, and in full appreciation of the good results which have been obtained by the use of such grafts. There is abundant demand for the use of both pedicle flaps and detached, full-thickness skin grafts. However, it is important to appreciate the possibilities of both of these forms of treatment and to select the one which is best for each individual case.

Case I.—A boy, aged eleven and a half years, had been scalded when one and three-quarters years of age. His neck had been persistently massaged and rubbed with oil during the intervening years. The cicatricial skin was thin and pliable, but it was short from above down, as is characteristic of these cicatrices, although laterally there was more than enough skin to encircle the neck. The cicatrix stood out from the neck almost like a guy rope, extending from the outer side of the chin to the chest wall. A single operation by the cross-flap method relieved the greater part of the contracture, allowing free motion of the head and neck, and relieving the downward traction on the right side of the mouth. The result of this single operation was so good that we could never obtain permission for further adjustment.

This case illustrates two important factors: 1. The pliability and absence of thickening which may come in cicatrical skin. The original wound must have been deep, else there would not have been so much contracture. Yet, during this long period the cicatricial skin had become thin and pliable and well suited for

FIG. 1 FIG. 2

FIG. 1 (Case I).—Cicatricial contraction of neck, the result of scalding in infancy.
FIG. 2. Same patient as Fig. 1, showing transverse fulness and longitudinal shortening of the cicatricial skin. The pencils show the outline of the neck.

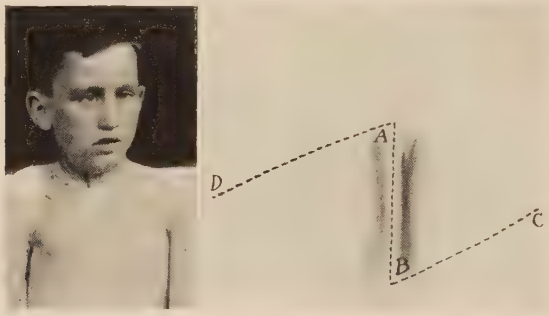

FIG. 3 FIG. 4

FIG. 3.—Favorable result of a single operation by the crossover-flap method.
FIG. 4.—Formation of flaps for use in the crossover method. *B* is carried upward and across toward *D*. *A* is carried downward and across toward *C*, thus lessening the transverse fulness, relaxing the contracting tissue and putting the width of two flaps into the contracted area.

use in flap formation. This is important, since some very skilful surgeons have advocated the ablation of the cicatricial skin as a preparation for flap implantation.

2. The cross-over flaps are very useful where there is shortening in one direction and undue fulness in the other. Their use

was first called to my attention by my colleague, Dr. Frank S. Mathews. I was treating cases of contracture from burns by full-thickness skin grafts with good success, and presented a paper on the subject before the New York Surgical Society; but I found that he was getting prompter results from these cross-over flaps and I have used them more or less ever since. Dr. Mathews devised the plan independently, but I learn that Piechard had used a somewhat similar plan in the axilla. (Fig. 4 illustrates the method.)

FIG. 5 FIG. 6

Case II.—Cicatricial contraction from burn. Front and side views.

CASE II.—A boy, aged six years, was brought to the Roosevelt Hospital six months after receiving a severe burn. The extensive mass of scar tissue is shown in Figs. 5 and 6. It extended from the entire lower part of the face downward onto the chest and drew the chin down to the manubrium and held it there so firmly that the head could not be extended without opening the mouth. Most of the scar tissue was thick. There were longitudinal bands in it. In the middle there was an ulcer 1 inch in diameter. On pinching the skin one found the usual excess of lateral tissue and an extreme shortage of longitudinal tissue. It was evident, however, that this cicatricial skin had some possibilities for usefulness.

The first operation consisted in the formation of cross-over flaps in the front of the neck and in the sliding upward of a pedicle flap from the chest so as to partially fill in the defect thus made.

Seven weeks later a long transverse incision was made across the neck so as to leave an apron of cicatricial skin beneath the chin and body of the jaw. This was pushed up beneath the jaw and stitched

FIG. 7 FIG. 8

FIG. 7 (Case II).—Diagram of flaps used in the first operation.
FIG. 8.—Appearance of patient after the first operation.

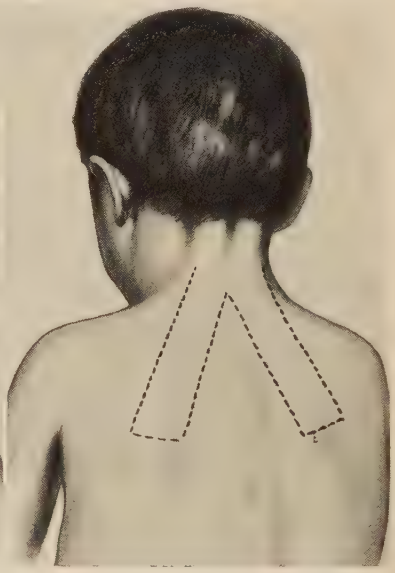

FIG. 9 FIG. 10

FIG. 9 (Case II).—Diagram of flap used in second operation.
FIG. 10.—Diagram of pedicled flaps taken from the back and hinged around the neck.

FIG. 11 (Case II).—Appearance after the second operation.

FIG. 12

FIG. 13

FIG. 12 (Case II).—Appearance after the healing of the pedicled flaps.

FIG. 13.—Appearance after permitting the flaps to adjust themselves for nine months, and then dissecting out subcutaneous fat and thickened connective tissue from beneath the chin.

there. Pedicle flaps were then swung upward again from the chest. These two operations gave very definite improvement in the condition.

After another seven weeks a cut was made through the skin around the entire left side of the neck and carried across the median line in front, permitting extension of the head to the normal degree. A flap, 5 by $2\frac{1}{2}$ inches in size, with its pedicle at the nape of the neck, was then raised from the left scapular region and swung around into the defect. This flap held well and gave great improvement. Four weeks later a similar procedure was adopted on the right side. This also held well. After the healing there was abundant cutaneous tissue in the front part of the neck, but it showed more thickening than was desired, and there was a tendency to form a median longitudinal band. The boy

FIG. 14 (Case II).—Illustrating the amount of extension of the head and the outline of the neck after the operations previously described.

was therefore sent home for nine months to learn how much adaptability the newly adjusted tissue would have.

An opening was then made under the chin and subcutaneous fat and thickened tissue were removed in considerable amount, and flaps were readjusted so as to relieve the small longitudinal median band. This gave great improvement.

He was again seen a year later. The result was wonderfully good, as is seen in the accompanying photographs. There was a slight tendency to ulceration in the median line, hence another small plastic operation was done to remedy this defect.

This patient was notable in many respects, not the least of which was the willingness of himself and his relatives to come and go as desired

so that the ultimate adjustment of tissues could be obtained. A very small part of the original scar tissue was sacrificed. The skin thinned out with time and adjusted itself to the underlying tissues so that the defect is of little importance. He has normal motion of his head, is a particularly good-looking child and is able to go about his play and his school duties without embarrassment, and it is fair to believe that his injury will not form a material handicap in his later career.

Fig. 15

Fig. 16

Fig. 15 (Case II).—Appearance while a small median band remained which was irritated by the clothing.

Fig. 16.—Appearance on discharge from the hospital.

Case III.—At this point Fig. 6 should be inspected. It shows an extreme degree of the longitudinal shortening and the transverse fulness. A sheet of cicatricial tissue extends from the border of the jaw to the chest, obliterating the outline of the neck itself. This case was particularly suitable for the use of pedicle flaps hinged forward from the scapular region.

At the present time each surgeon, in dealing with this type of contracture, must carefully consider the relative merits of full-thickness skin grafts and of pedicle flaps. Of late there has been much progress in the use of the full-thickness grafts and both their advantages and their disadvantages are being better appreciated. Blair makes the following judicial and satisfactory statement:

"Pedicle flaps when properly handled are more certain of complete success, and, if taken from the neighborhood, are apt to have a better color.

"The full-thickness graft is somewhat uncertain of result, is exacting in its technic, requires three weeks before it can be considered safe and in children and young adults may develop red-corded scars months after an apparently perfect take, yet it has a place in surgery that is not as well filled by either of the two preceding" (pedicle flaps and Thiersch grafts).

FIG. 17 (Case III).—Showing a very broad, shortened, cicatricial area. A case particularly well adapted for the use of pedicled grafts hinged from the back.

Most surgeons will use pedicle flaps when there is available tissue for their formation. When such tissue is not available, full-thickness skin grafts will be used. For instance, Blair describes a patient in whom the cicatrix pulled the lower lip so far downward as to evert it and expose a large area of its mucous membrane. Three full-thickness skin grafts were applied at three different operations. Manifestly there was not enough available skin to form efficient pedicle flaps for this case, and Dr. Blair and the patient are to be congratulated on the good result obtained under these conditions.

CONGENITAL CLEFT LIP AND PALATE

A Muscle-theory Repair of the Lip Cleft

By H. P. RITCHIE, M.D.
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Repair of a lip cleft on the muscle theory is based on a general surgical operation, the union of a cleft sphincter muscle. It is not my purpose to propose a new method of closure, but to discuss principles and procedures involved.

The terms here used are those of the classification which John Staige Davis and I offered a few years past. The suggestion was made of a very simple and rational change in the concept of the general subject. The literature has been built upon the original idea: A dual point of view, indicated by the title, "Hare Lip and Cleft Palate." The change suggested is that the many problems be rediscussed upon the basis of a *series of congenital clefts.* The essential points of the plan are that the complete palate is resolved into its component parts and the term "hare lip," long recognized as an inapt if not misleading term, is erased. Each cleft is a result of failure in the same embryonal closure; no one to be emphasized over another, except as it appears of more importance in a given case.

The proposal offers an opportunity of describing separately the lip cleft, the alveolar-process cleft, the hard-palate cleft and the soft-palate cleft, not only as to form, but what is of equal, even more, importance, the degree of the cleft. I believe that many problems now under rigorous debate can be settled on the basis of degree. I am impressed by the observation that controversy as to procedure in this surgical field is often caused by a loose and indirect terminology, and that the use of common descriptive terms would greatly obviate this tendency. Thus by our plan of record, the arrangement of the tissues may be exactly recorded in direct, positive and unvarying terms.

FIG. 1.—Two hare lip operations are here compared. Primary results were entirely satisfactory. One grows beautifully, the other with deformity. My opinion is that in one the tissues were replaced in a way normal to that baby, in the other the effort failed.

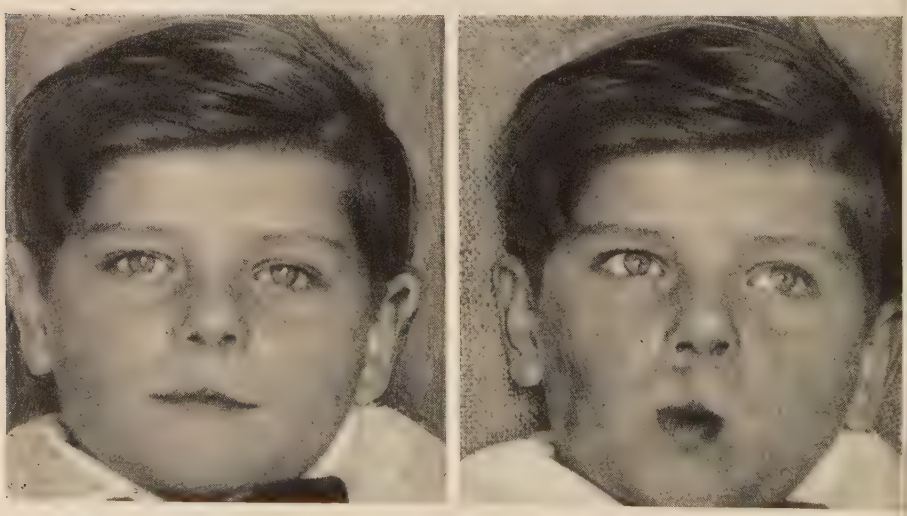

FIG. 2.—There are several cases with splendid cosmetic results which show varying degrees of distortion on motion of the lip. This is my principal reason for approaching the problem of lip repair from the angle of function.

It requires only a cursory examination to prove that each cleft involves different tissues which require consideration of appropriate surgical principles. The procedure in closing each cleft must conform to the character of the tissues and, since these vary, the technical phase must be approached on the plan of

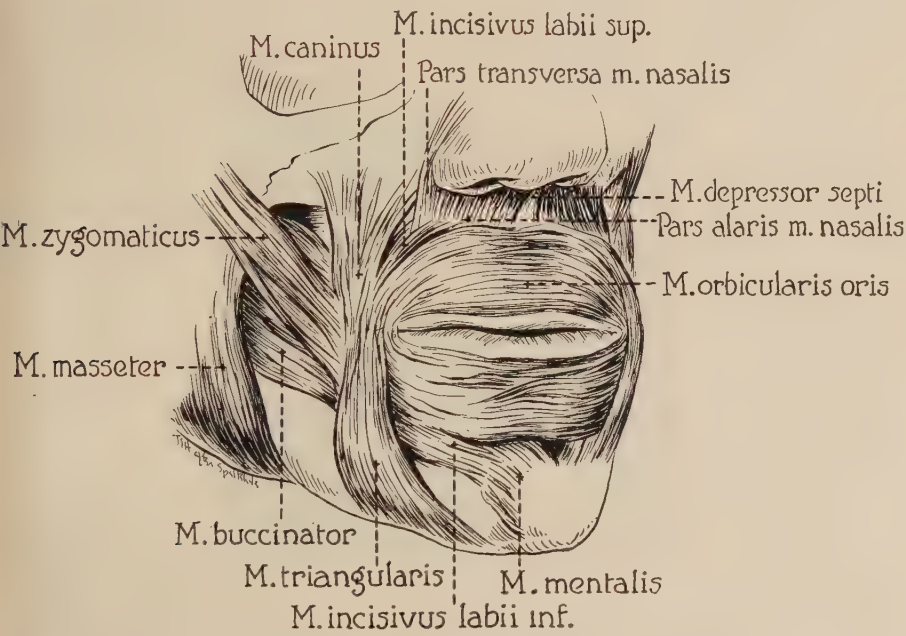

FIG. 3.—The muscle-theory repair of cleft lip is based upon the study of anatomy of the lip in the adult. The active tissue in this structure is the orbicularis oris muscle. It is so arranged as to permit of special motions, but its composite action is that of a sphincter, puckering the tissues as in whistling. By reason of its position it must be further considered. Radiating from it are several muscles noted in the drawing. Thus it must be considered the key ring of the expression muscles of the face and is the medium of contact between the right and left groups.

separate clefts, the methods of repair to be determined independently.

The purely surgical problem, judgment as to time, combination and sequence of operations now becomes widely broadened. The hare lip and cleft palate idea is too narrow. It fosters the use of special routine methods for the repair of either one or the other.

The series proposal gives to this field the viewpoint of general surgery—selection of procedures to fit the case.

THE THEORY. Of the series, the lip cleft has always been recognized as a problem differing from those of the palate. The repair has been approached as a special hare-lip operation. There have been many suggestions as to straight, curved or angulated lines of incisions, cross-cuts, flaps, tissues mobilized out of posi-

FIG. 4.—If the premises stated in Fig. 3 are correct, it may be argued, and I believe concluded, that the purpose in lip repair is the exact apposition of this contractile tissue. The above picture is worthy of close study because it represents the natural plan and procedure. This boy is a prealveolar cleft, the lip being the only one of the series in this group. Nature failed only at the mucous membrane body of the muscle. But in making contact the suture line on the skin was left. On the lip the line hugs the left border of the philtrum and continues up into the floor of the nostril. I interpret this case as a wonderful model for surgical repair and it indicates lateral approximation of the muscle.

tion to guard against stitch tension, tissues reflected out of position to prevent contracture and many follow-up plans of support. The purpose of the effort is to make the lip look as normal as possible, thus elevating in importance the cosmetic result. At least that is what I have been trying to do. If it is a unilateral cleft I try to make the defective side look like the normal; if a bilateral, to make the two look alike.

In the few years of my interest in this field of surgery I have increasingly felt the need of a more positive purpose in the operation, a motive more direct than looks. The idea occurred that if the problem was studied from the aspect of function, a greater assurance of a satisfactory, possibly perfect end-result would follow. So I offer suggestions for the repair of the lip based on the idea that restoration of normal function is the primary object, believing that proper cosmetic result will immediately follow the operation or can be accomplished at a later date.

FIG. 5.—Under anesthesia the outlines of the muscle are not evident except by some surface markings, nor is it visualized during dissection. It must be there because in the adult a powerful band is present. I thought if this tissue could be contracted its exact outlines could be determined.

The variation in my hare-lip operations is too wide, ranging from beautiful results to very unsatisfactory ones. Cases which started well are not growing normally; cases starting poorly are improving each year. There are quite a number of children with splendid cosmetic results, who show definite distortion of the lip on motion, both in speech and expression

Some explanation of this variation other than operative failure must be made. The question naturally arose whether in the good lips I had not replaced the cleft tissues in a way normal to that baby. This thought led to the question whether there was not some characteristic arrangement in each baby's lip that would

lead to the replacement of tissues out of contact in a way normal to that individual. This appears as a proposition leading to an ideal. But attention is called to the word "individual." If one goes through a babies' ward, critically records the various contours of nostrils, the long, short, thin, thick lips, with varying degrees

FIG. 6.—The agent used to contract the tissues at operation. This is a one dry-celled faradic battery prepared as stated in the paper.

of mucous membrane eversion, then our present plan of repair, a set procedure plus the personal interpretation of the operator, must yield in the end some degree of variation. Though cases appear similar at this early age, no one can visualize their aspect in adult life. So many writers have called attention to the fact

that it is not the immediate but the end-result that will prove our work. In other words, the time element must enter argument and effort. Proper repair will depend upon normal replacement plus the years of growth.

Therefore an answer to the ideal proposition must be sought in the study of the lip in the adult. This reveals that the body of the lip is mainly composed of a structure called the orbicularis oris muscle. All other tissues are mere coverings for this muscle. It is

FIG. 7.—Under stimulation the muscle contracts to almost a right angle. Out on the mucous membrane the lower border is shown. This point is indicated by the knife point.

the only active tissue in the lip and, being muscle, conforms to the behavior of similar tissue elsewhere in the body, hypertrophies on use, atrophies on disuse and normal action depends upon proper origin and attachment. As a muscle body it varies in size, distribution, power and function, as do all muscles in different individuals. Intrinsically, it is a sphincter at least in its lower fibers, puckering the tissues as in the act of whistling. It is important to remember that its use is an essential in proper speech. But by its position and association it must be further considered.

Radiating from it or into it are all the muscles of the face, a group which may be called expression muscles; the zygomaticus, ala depressors, buccinator, etc. Every single or group action of these indirectly affects the sphincter, any action of which must work the reverse. In other words, the orbicularis oris is not only a pucker muscle, but is in effect the key-ring of all the expression muscles of the face. It is the main medium of contact between the right and left facial groups.

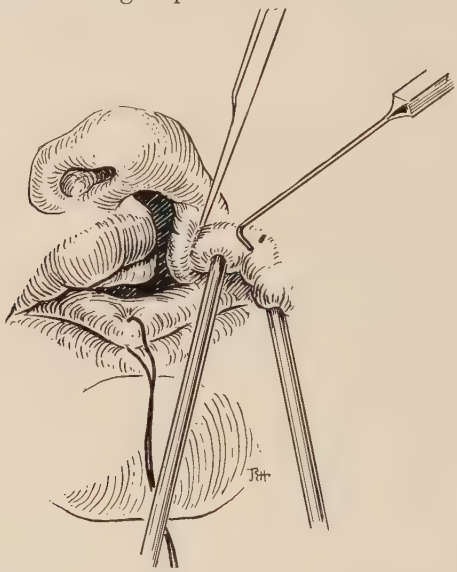

Fig. 8.—The lip is reflected upward and outward and the points of the battery again applied. This maneuver develops a most important point, where the wing of the nose, the alveolar process and the muscle meet. To properly expose the latter an incision is required. This varies (in different babies) in length, depth and inclination. How much of the skin is involved in the cut I am uncertain, but I am sure that a nick should be made.

The above is a statement of anatomic facts, long known and repeatedly demonstrated. Carrying this established picture of the adult to the congenitally cleft lip, it appears a reasonable conclusion that the motive in the repair of the lip is the exact approximation and union of the cleft body of this muscle. The opportunity for normal growth and the permanency of any associated plastic repair will depend upon the precision with which this is accomplished.

THE OPERATION ON CLEFT LIP UNILATERAL. That this problem has not been generally approached from the angle of function is due to the fact that in the sleeping, inactive infant the contractile quality of this tissue is not demonstrated; its outlines not shown except possibly by some surface markings, and during the dissection it is not visualized as muscle. But it must be there, because in the adult the powerful band of the lip is present.

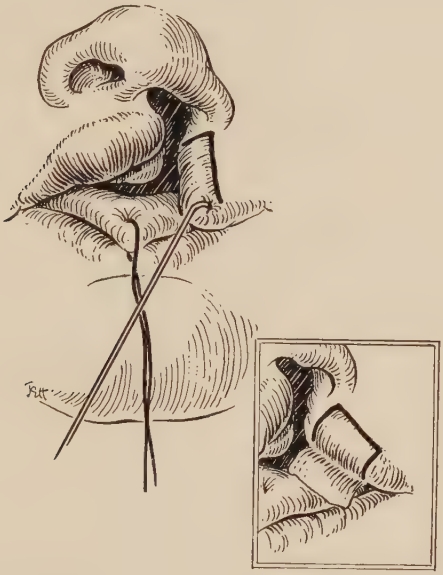

FIG. 9.—With the lip pulled out on the stretch, a perfectly straight line incision is made connecting the point on the skin above to the point on the mucous membrane below. While on the stretch the mucous membrane is carefully dissected backward, as a flap, as shown in the insert. This exposes the contractile tissue which was determined under contraction. The same procedure is followed on the other side.

The idea came that if it were possible to contract this tissue at operation four cardinal points could be obtained; the upper and lower margins on each side, and that exact lateral approximation would then lead to the union in a way normal to that baby.

I remembered the muscle-juggling experiments of our laboratory days with the faradic current; combed the market to find a cheap one-celled dry battery. The therapeutic attachments were dis-

connected and two 3-inch long copper wire points were made for the distal ends of the cords for muscle stimulation. The proximal cord ends were put in No. 1 and No. 3 holes of the battery and the rheostat pulled half-way out, thus obtaining an agent to contract this tissue and show its exact outlines.

To clearly describe the steps of an operation is a most difficult effort. I have followed the scheme of steps, herein described, as being the most direct and concise plan that I know:

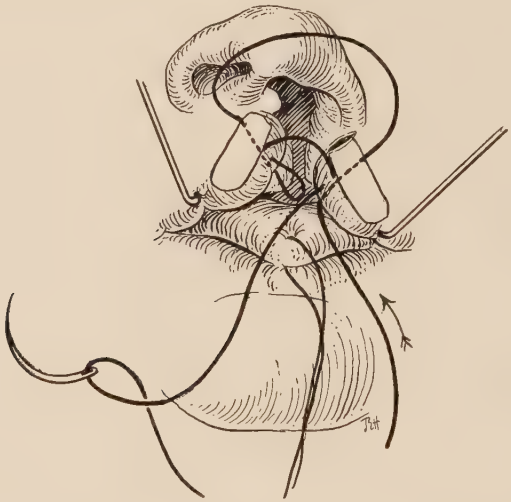

FIG. 10.—Any mobilization of the ala should be done after the denudation of the short side. When this procedure is done, two surfaces appear that are equal in length and width, in perfect position for lateral union. I am unable to make them as perfect as shown, but that is my purpose. The stitch shown is most important and should include the very upper fibers of the muscle. It is this stitch that determines the degree of rotation of the ala.

1. On the short side one copper wire point is placed at the angle of lip, the other near the cleft margin, both held firmly on to the mucous membrane, and the switch swung on. Under stimulation the left muscle jumps up like a biceps under contraction and the usual curve of the lip contracts almost to a right angle. The cleft margin looks like a wall, the width of which is the thickness of the lip of that baby at the time of the operation. Out on the lip quite frequently a pit occurs, due, in my opinion, to contrac-

tion of the real sphincter fibers. A nick in the mucous membrane is made at this point to represent the lower margin of the contractile tissue.

2. The lip is reflected backward with a single tenaculum or a stay stitch and the points again applied. The muscle again contracts, revealing the upper margin. In order to expose this border an incision is required which, I believe, varies in inclination and degree in different babies. I am uncertain how much of the skin is included in the cut, but I feel sure that at least a nick should be made.

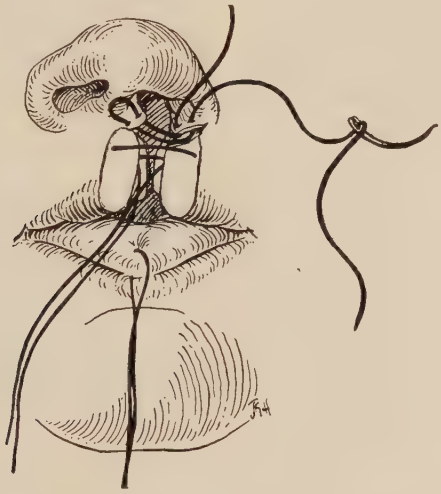

FIG. 11.—The top muscle stitch is pulled down, and a lateral mattress placed in the denuded area of the ala and the epithelial reflection at the base of the columella. This reflection is up in the nostril. In the drawing it looks as though it was upon the lip. In all adults, the floor of the nostril has a length and is nearly at right angles to the lip. Two other muscle stitches are placed, and the skin incision closed.

3. The lip is allowed to fall into place. By means of a tenaculum or stay stitch the lip is stretched. The two points, one above on the skin, the other below on mucous membrane, are connected by a perfectly straight incision, taking a thin shaving of skin.

4. While the lip is still on the stretch, by close and careful dissection the mucous membrane is dissected back as a flap, thus

exposing the sphincter in perfect position for lateral approximation to the opposite side of the cleft.[1]

5. The denuded lip is packed and pressure applied to the bleeding surfaces.

6. The same procedures are used for the long side, with two additions. The frenum is carefully preserved, as we are trying to make a normal lip. At the base of the columella, extending into the cleft nostril, is an epithelial structure which I have always used as the inner half of the floor of the nostril. Under contrac-

FIG. 12.—Lateral approximation of a cleft sphincter muscle is the expression I have used. I am not sure that this is correct. So many cases show an oblique suture line. It must be remembered that the obicularis oris is formed on a curve. A cleft on one side of the exact middle of the curve would be on an oblique plane. Possibly, end-to-end union of a cleft sphincter is more exact.

tion it is necessary to raise this reflection in order to fully expose the upper border of the muscle, proving that what I had done indirectly now becomes a direct step.

[1] At this point the ala should be brought forward to a level of the base of the columella. Whether the lip is a prealveolar cleft or an alveolar cleft, whether the process cleft is to be closed by the lip or has been closed by mechanical measures either immediately preceding the lip operation or at a remote period, the ala is quite constantly depressed and must be moved to a plane conforming laterally to the normal nostril. This should be done after the muscle is exposed, because should it be attempted before denudation the exact outlines of the orbicularis and its normal relation to the underlying process may be lost.

FIG. 13.—A group of cases of cleft lip unilateral showing results of repair based upon the muscle theory. All of these cases look very well on front and side view. They are taken in position to show and in some of them exaggerate any deviation from the normal. Included are two of the worst results. They were selected to show the greatest variation in contour of the nostril, length of lip, and eversion of mucous membrane. I have done many more than here pictured and can say that no two babies look alike. How they will appear in adult life I do not know. It is for this reason that I am so specific in offering these suggestions only as a theory.

7. A hook is fixed in the denuded muscle which is pulled out as is done in complete laceration of the perineum. A stitch is

FIG. 14.—I have had only one median cleft. It is a perfect example of lateral approximation because the cleft is at the top of the curve.

FIG. 15.—Only one cleft lip bilateral has appeared during these studies of the muscle-theory. But it seems to work out on the plans described in the paper.

inserted exactly into the upper fibers of the short side and across to the long side, another in the middle and a third in the lower

sphincter group. All this is for the purpose of opposing the cleft muscle from side to side without distortion of it in any way. As the top stitch is tightened the ala swings in to meet the epithelial reflection, mentioned in 6. At this time any plastic work on the floor of the nostril and ala can be done. If the muscle theory holds, this should not be necessary, but I have so far found it advisable to at least make the raw margins more exact, but believe that as little plastic work on the nostril as possible should be done. A lateral mattress stitch is then placed to make a floor to the nostril. This floor is a definite anatomic entity, observable in all normal individuals, but seldom mentioned in any of the hare-lip operations.

Fig. 16.—The principle applied as a secondary repair.

Then the skin stitches are placed and the muscle stitches tied underneath, thus accomplishing the principle of a two-series suture line, which I believe is a most important procedure in all plastic repair of the face and neck.

The muscle stitch represented in the drawing is the vertical mattress which is a nonconstructing, broadly opposing stitch, and therefore applied to muscle tissue. These are placed in from the mucous membrane side. By so doing the best chance of completely grasping the muscle fibers is accomplished. I have

thought of using the figure-of-eight stitch. I have been using this stitch in the soft palate for three years or more, with the greatest satisfaction. It has been of general use in my laparotomy wounds, breast flaps, etc., and is of great value because it combines approximation with a tension stitch and is indicated in the lip and soft palate because we have in both locations active tissue, which, once united, assumes some degree of contraction, and pull against mere coaptation stitches immediately begins. Lateral approximation of cleft contractile tissue without distortion or displacement is the basic principle upon which the above plans are developed.

Fig. 17.—This man of thirty years seems to answer the question raised in the paper: Is there muscle in the prolabium? Two mounds of muscle appear on either side, with a thin skin and mucous membrane curtain between. When the muscle—perfectly possible at this date—was brought together underneath, function of the lip and coördination of the expression group were obtained.

The Median Cleft. I have concentrated upon the unilateral cleft lip because in the study of incidence of form and degree, according to the above-mentioned classification, this condition occurs in about 70 per cent of cases. It is a most frequent problem, not only in the alveolar-cleft group, but in the group of prealveolar clefts. But there are included in this general question of the lip two other forms, namely, the median and bilateral. I have seen only one median lip cleft, which however was so perfect that the frenum was symmetrically divided and attached to each of the cleft processes. I mention this because there was in this case no question of mobilization of tissues or any thought

of rearrangement, but only definite, exact, lateral union. This resulted, of course, in a perfectly natural lip. I report it because it is the best example of the principle of lip repair which I am endeavoring to promulgate, *i. e.*, lateral approximation of the muscle structure.

THE BILATERAL CLEFT. To apply this effort to the bilateral cleft lip difficulty is immediately encountered, and can only be solved by answering several questions which I have raised and presented to several consultants. The first and most important of these from a surgical standpoint is the presence or absence of muscle structure in the prolabium. Dr. R. E. Scammon, professor of embryology at the University of Minnesota, thinks there is no muscle here unless it comes down from the frontalis, which is a long journey. I have been unable to demonstrate it with the battery in the complete cleft, but if one side is incomplete then contraction takes place. I have, in one case only, studied the removed rim of mucous membrane histologically, and this contained no muscle. If muscle is not in the prolabium of the bilateral cleft, how does it get there in the normal lip? Dr. J. E. Thompson, of Galveston, referred me to Keith's *Embryology*, where an explanation is made. This refers to an embryonal movement of muscle in its distribution expressed by the word migration. Whether this is a proven fact or not, it offers the most rational explanation, because in normal lips the contractile tissue is a band formed *underneath* the prolabium which becomes the philtrum. From a surgical viewpoint of normal repair, then, the orbicularis oris is mesially cleft with equal groups of fibers on either side.

If the muscle theory holds, plans must be made to bring the cleft ends together in the midline on the mucous membrane side. It appears impossible to unite the band mesially in its entirety, but it is an established fact that if a muscle is anchored to another structure and heals it will assume function in its new position. This may be made use of for the upper and middle sections of the muscle which are sewn to the prolabium. In order to obtain real function, the sphincter, that is, the lower group, must be approximated mesially and behind, thus building a lip long on the mucous membrane, while the length of the skin side is determined by the extent of the prolabium.

In support of these arguments, I present illustrations of three cases: One, a primary effort; another, a secondary repair of a

boy, who had no power of expression in the lip until the muscle was found and mesially sewn together with the result shown in the picture taken two weeks following repair; a third, a man, aged twenty-eight years, lacking not only function of the lip, but all power of expression, assuming that blank, mournful look which so many cases of imperfect lip repair have. The picture of the adult appeals to me as most conclusive in support of the above arguments. Time and effort had so developed these groups of muscle tissue that they become very evident on inspection. This lip was only a curtain. As soon as the muscle was approximated the whole outlook of this man's life was changed, and he then had control not only of the lip but of the right and left muscle groups of expression.

The Histology of the Lip. The arguments of the muscle theory are most alluring and are so direct that I have been led to wonder why it was possible to have approached this repair by so many and various plans. I think the explanation lies in the study of the histology of this muscle. This reveals a most complicated picture of muscle bundles, varying in size and distribution, connective tissue, glands and fat. Certain bundles appear to interdigitate Nature's plan to permit of some special action of the lip other than sphincteric action.

Conclusion. Whatever plan is adopted, some kind of lip can always be made. The main point is whether the procedure results in the replacement of tissues in a way normal to the individual baby. There are three procedures in the literature that nearly meet the muscle theory: The Mirault method, in which the undercutting of the ala most nearly resembles the incision, for the exposure of the upper margin of the muscle; the Rose operation, which symmetrically prepares for lateral union but, I believe, sacrifices tissue unnecessarily; the use of the Thompson calipers, which, by the exact measurement of the normal and defective sides, offers the greatest chance of all the recent methods of exposing the muscle ends.

In conclusion, I wish to again emphasize that the above is a theory, and I do not believe that anyone with an established and satisfactory method should change his technic. The battery may be used to check the lip and determine the value of the steps used. What I see in the theory is that it establishes a definite principle of function and thus institutes a change from indirect to direct surgical methods.

PLASTIC SURGERY: REMOVAL OF SCARS BY STAGES

An Open Operation for Extensive Laceration of the Anal Sphincter; the Kondoleon Operation for Elephantiasis

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Simple surgical procedures are often used and forgotten, and for this reason I wish to report in this symposium on plastic surgery two simple but useful surgical measures which, no doubt, have been used before but to which I am unable to find references. I also wish to discuss certain modifications of the Kondoleon operation which have proved valuable in improving the end-results in the treatment of elephantiasis.

Removal of Scars by Stages. The skin, probably on account of its elastic fibers, easily stretches when put under tension. We see examples of this in large tumors, such as goiters and in cases of elephantiasis after the edema has been reduced by bandages and rest in bed. It is possible to take advantage of this characteristic of the skin and to remove scars situated in certain regions where it would be inadvisable to use grafts, in stages, allowing the skin after each operation to stretch and again become loose and movable. From the appearance of scars following skin-grafts, from the color of the skin in these areas, or from the border of the graft when thick grafts have been applied, one can usually tell that grafts have been used. The appearance of the scar for which the original operation was performed may have been greatly improved, but the result is not always that desired by the operator.

Case I.—The patient was a woman, aged thirty years, who had been affected with goiter since the age of sixteen years. From many expo-

sures to the roentgen-ray in the treatment of goiter, a superficial burn resulted and the scar which followed involved almost the entire anterior surface of the neck, from the hyoid bone nearly to the clavicle. Its appearance was so unsightly that the young woman was quite sensitive about it, and to hide it wore at all times a scarf or a high collar. She had repeatedly consulted surgeons in the hope that it could be removed, but had always been advised against operation, since they felt that the skin-graft which would be necessary after removal of the scar would be almost as unsightly as the burn.

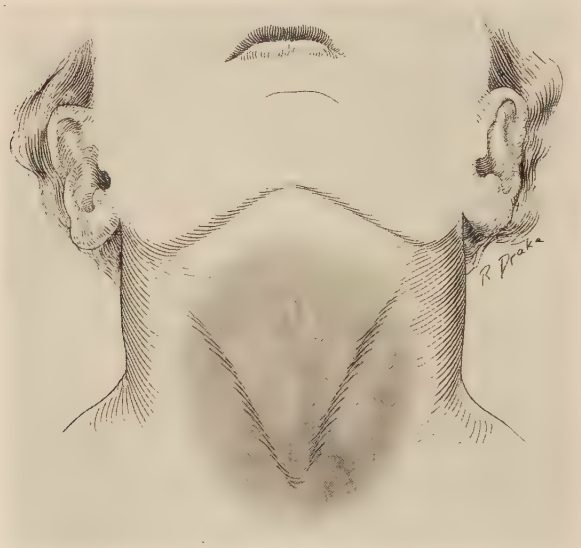

Fig. 1.—Appearance of roentgen-ray burn before operation.

Examination showed the scar of a superficial burn with movable skin which was unattached to the deeper tissues. It was quite red in appearance and showed many large, dilated capillaries. Apparently nothing short of complete excision of the burn would improve the appearance of the neck. It was accordingly suggested that the burn be removed by stages to which method of treatment the patient gladly consented.

As much of the skin as could be removed and still allow closure of the edges of the wound without tension was removed. The skin and superficial fat only were removed and special effort was made to avoid undermining the skin in order to prevent adhesions between it and the deeper structures which would later prevent the skin being stretched by massage, and so forth. It was possible in this way to remove about

one-third of the scar. Following the operation the patient stretched the skin by movements of the neck and by massage. On her return, six months later, the skin was as loose and pliable as it was at the first

FIG. 2.—First stage of the operation. A small portion of the burned area excised and the wound closed.

FIG. 3.—Second stage of operation (repetition of first stage).

operation and the scar was only two-thirds as large. A similar operation was then performed, to remove about one-half of the remainder of the scar. The patient returned again after six months and, at a

third operation, the remaining portion of the scar was removed. Now only a single line may be seen. In this way the skin was removed from almost the entire anterior surface of the neck without a graft (Figs. 1, 2, 3 and 4).

Fig. 4.—Third stage of operation. Remainder of burned area excised and wound closed.

I have since employed the same method in removing an ugly scar from the anterolateral surface of the neck. The patient, a young woman, had suffered a severe superficial burn following the application of a strong solution of iodin. The results of the operation were excellent.

An Open Operation for Extensive Laceration of the Anal Sphincter. In the ordinary case in which a primary operation is performed for a tear of the external sphincter ani the operation almost universally described in textbooks is quite satisfactory and is usually followed by good results. However, an occasional case is seen in which practically the entire external sphincter has been destroyed, usually either by an infection, as is seen in perirectal abscesses, or as a result of an operation for rectal fistula.

In the type of case which I am attempting to describe the sphincter has often been largely destroyed and the nerves supplying it usually impaired or destroyed. In many such cases it is almost impossible to draw the ends of the sphincter together without such tension as to prevent satisfactory healing. The anus, too, in such cases, is necessarily so tightly closed in bringing the

ends of the sphincter muscle together that too small an opening is left and the muscle ends usually separate during the first act of defecation. Should satisfactory healing occur, the normal control of the muscle is not regained, and the anus cannot be closed even by voluntary efforts.

For some years I have, in treating anal fistula, divided the tissues anterior to the fistulous tract and severed the external sphincter over a probe. In order to prevent wide separation of the sphincter ends and still be able to pack the fistulous tract with gauze, so as to allow the wound to heal from the bottom by granulation, I have sutured the cut ends of the external sphincter muscles to the internal sphincter (levator ani muscle). An operation of this sort has usually cured the fistula and at the same time has established excellent control. The effect on control of bowel movements has been so satisfactory that I have extended the use of the operation and now follow practically the same method in operating for complete lacerations of the perineum and in repairing the anal sphincter when it has become incompetent from any cause.

Technic. A curved transverse incision is made just above the anus or along the vaginocutaneous margin, and the levator ani muscle isolated. This muscle is sutured over the anterior rectal wall, as in an ordinary perineorrhaphy. The ends of the external sphincter, which are often widely separated, are isolated and sutured to the levator ani, the ends being brought as closely together as possible without too much tension. If sutured under too much tension satisfactory healing will not occur, as the tissues will slough and the sutures fail to hold. The ends of the muscle are usually brought close enough together to close the anus, so that it fits snugly around an index finger introduced into the rectum. The wound is left open and packed with iodoform gauze, as is done in operations for rectal fistula. The gauze is usually held in position by a suture placed through the edges of the skin and loosely tied in such a way that the edges of the skin are left wide open. After seven or eight days it may be removed and a healthy granulating surface usually results which slowly heals. The scar-tissue which forms in the healing process unites fairly satisfactorily the edges of the external sphincter. If the nerve supply to this muscle is uninjured the patient usually develops normal control; if it has been injured normal control will not be regained but the patient will usually be able to close the external sphincter voluntarily by

elevating the levator ani through efforts to close the vagina or to lift the rectum upward. Through this voluntary effort patients are usually able to close the anus fairly tight upon a finger introduced into the rectum and can usually keep the anus closed sufficiently long to control gases and feces until the toilet can be reached. The ability to close the anus by voluntary effort gives the patient great mental relief. The operation differs from that ordinarily performed for complete lacerations, as I have seen it described and carried out. In the common operation the ends of the sphincter are merely sutured together, often under great tension, without any attempt to suture them to the levator ani. The method I have described tends to give much better voluntary control, the only control that can be expected in certain cases in which the sphincter has largely disappeared and its nerve supply been injured (Figs. 5 and 6).

More satisfactory results are obtained when patients are given castor oil forty-eight hours previous to the operation and allowed only water, fruit juices and sugars after the oil has acted. I usually induce constipation by administering paregoric before the operation. This medication is also continued for about ten days after the operation, during which time patients are given only water, fruit juices and broth. After ten days oil enemas, consisting of 180 cc. of olive oil, are given by rectum morning and night, and a mild laxative, such as milk of magnesia, is administered several times a day until satisfactory bowel movements have occurred.

Certain Modifications of the Kondoleon Operation for Elephantiasis. These changes in the Kondoleon operation have been made after nearly ten years' experience with the operation and contribute to the success of the end-results. At the present time I feel that this operation is of much value in dealing with this disease. The operation cannot be considered one which will restore a perfectly normal limb, but as one through which the progress of this disease may be controlled and the size of the limb greatly diminished. Briefly, it may be stated that, although we see congenital elephantiasis, the so-called "idiopathic elephantiasis," which is usually seen in this country, is a sequel to partial or complete obstruction of the inguinal lymph nodes. Elephantiasis is always preceded by simple lymphedema which progresses until it gradually merges into elephantiasis. In lymphedema the

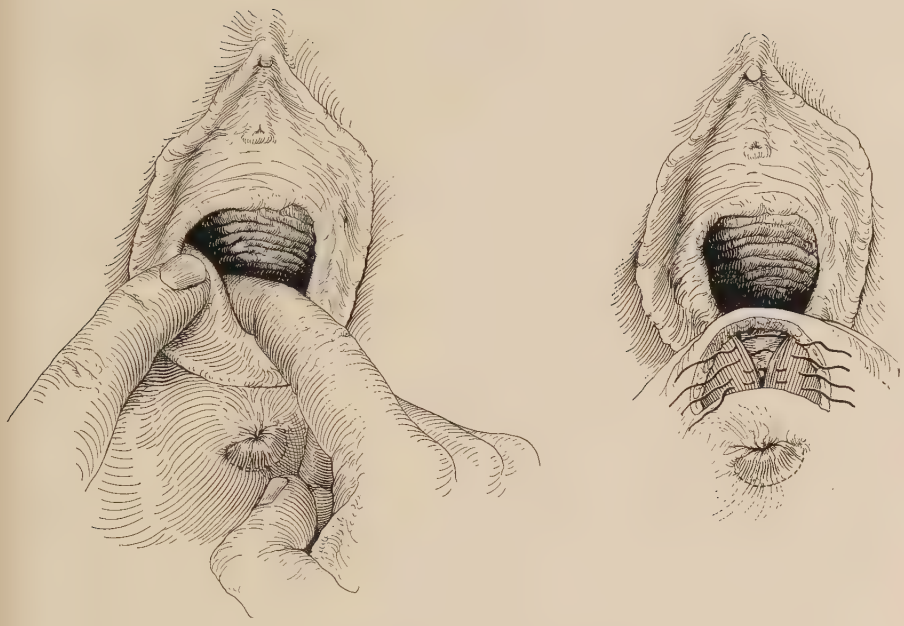

FIG. 5.—Exposure of levator ani muscles.

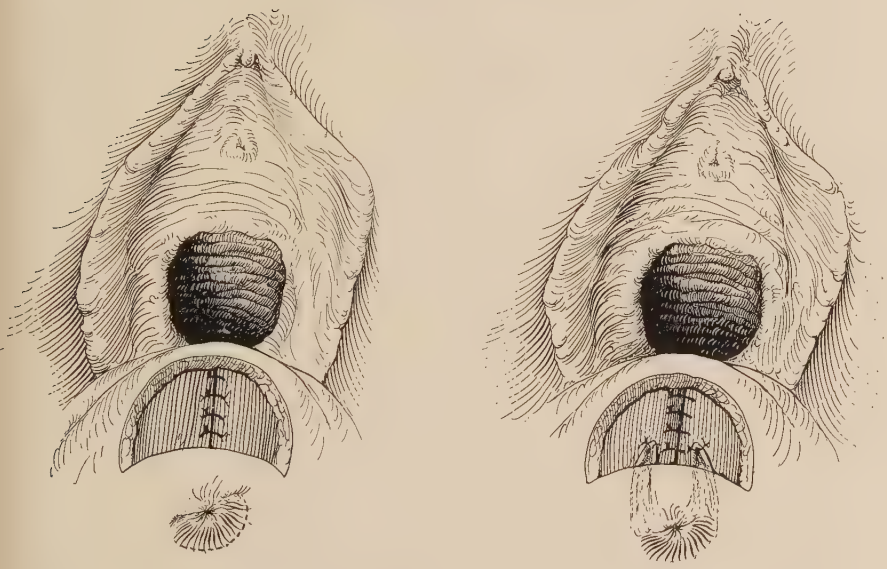

FIG. 6.—Levator ani muscles sutured together and remaining portion of anal sphincter sutured to them.

skin is thin, without the thickened dermis and aponeurosis and the large quantities of scar tissue seen in the subcutaneous tissues in true elephantiasis. The superficial lymphatic system alone is involved even in advanced cases of elephantiasis, and this system is definitely separated from the deep lymphatic system by the aponeurosis covering the muscles. The Kondoleon operation is performed with an idea of connecting these two systems by removing a large amount of this aponeurosis. At the same time, a large amount of the skin and subcutaneous fat is removed and the skin, with a small amount of the subcutaneous fat, is allowed to become attached to the muscles. When these raw surfaces are apposed new bloodvessels, new nerves and new lymphatics form, and the superficial lymphatic system is thus connected with the deep system.

I believe that the removal of large amounts of diseased tissue with its blocked lymphatics is one of the very important steps of the operation. Kanaval has called attention to the fact that this was suggested many years ago by Rogers of Milwaukee. Better results are obtained if much larger amounts of the aponeurosis are removed than first suggested by Kondoleon, and I feel that the more tissue removed the better the result. In order that large amounts of diseased tissue may be removed and the edges of the skin still be closed, patients should be carefully prepared for the operation by rest in bed for periods of from ten days to several weeks. During this time the limb to be operated on is kept elevated and firmly bandaged so that the fluids may be pressed out and the edema diminished. The bandages are changed as often as they become loose and the patients are made to get up and walk rapidly down the hospital halls for a few minutes several times each day, so that the blood-pressure may be kept as near normal as possible. At the present time a much more radical operation is performed than in the early cases.

Technic. A long, modified semi-elliptic incision, which includes the skin to be sacrificed, is made on one side of the affected limb. On the outer aspect of one of the lower extremities, for instance, this incision extends from the crest of the ilium to a point a little below the external malleolus. Then, in order to facilitate wide removal of the subcutaneous fat, the skin is reflected on each side of the incision for a distance of about 6 cm. The skin is then retracted and underneath each of the edges of reflected skin a long

incision is made through the subcutaneous fat down to and including the aponeurosis. These incisions are made almost parallel with the original skin incision. Included between them is a quadrilateral piece of subcutaneous fat and aponeurosis. The two incisions through the aponeurosis are then connected at their ends, so that the tissues to be removed are free, except for the attach-

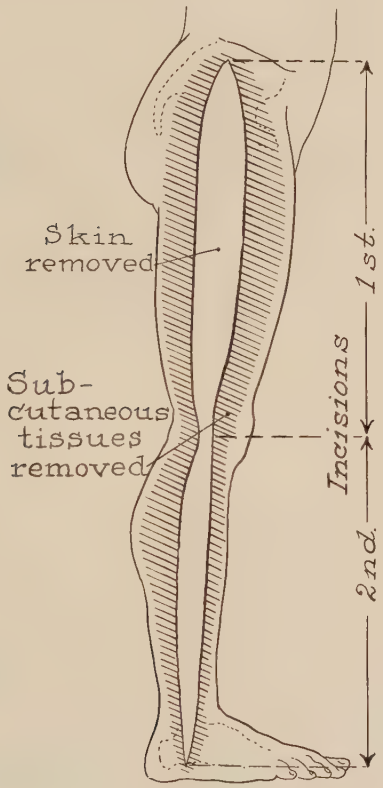

FIG. 7.—Strip outlined and undermined areas shaded.

ment of the aponeurosis to the underlying muscles. By traction on these tissues it is easy to dissect the aponeurosis from the muscles throughout the entire length of the limb and to remove in one long piece the skin, subcutaneous fat underneath it and aponeurosis. The skin with the small amount of subcutaneous fat is allowed to drop down on the muscles and the wound is closed with inter-

rupted silkworm sutures without drainage. It is usually necessary to perform a similar operation on the opposite side of the limb (Figs. 7, 8, 9, 10 and 11).

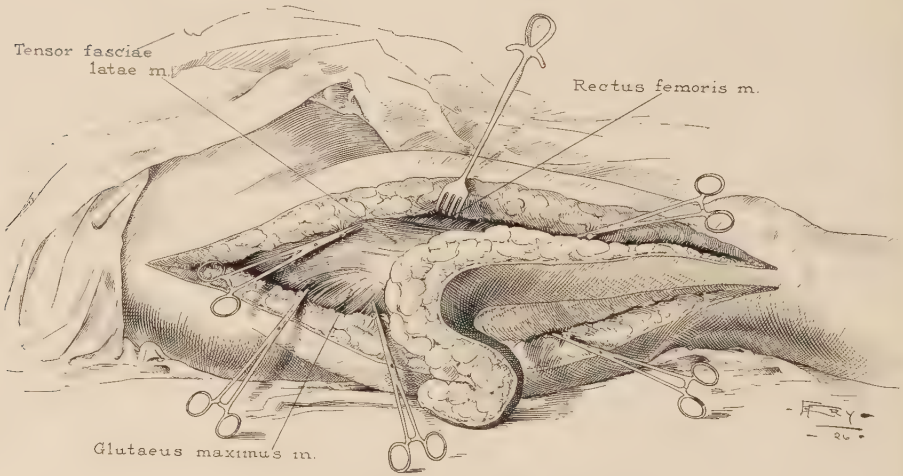

FIG. 8.—Strip nearly excised, vessels clamped.

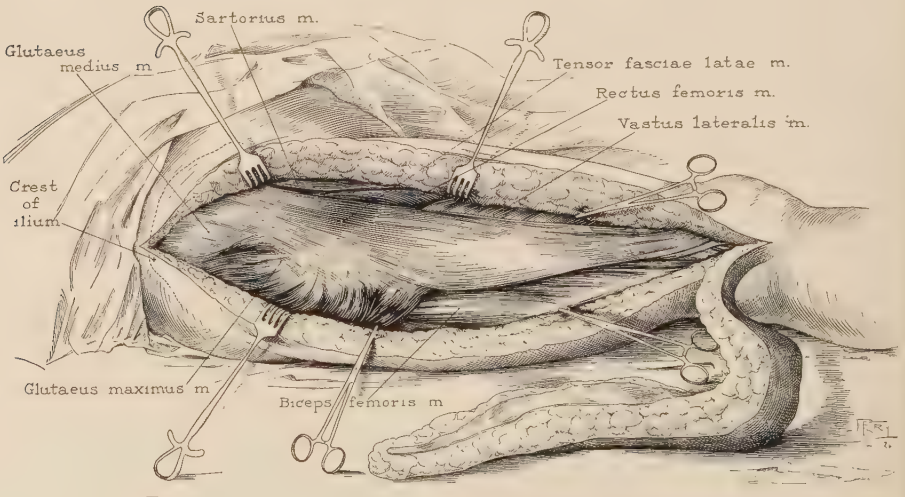

FIG. 9.—Strip from thigh excised with aponeurosis attached.

The operation is not devoid of danger, as very severe shock often follows a radical operation of this sort. Efforts are made to pre-

vent this, first, by keeping the blood-pressure up as near normal as possible during the preoperative period, and by the administra-

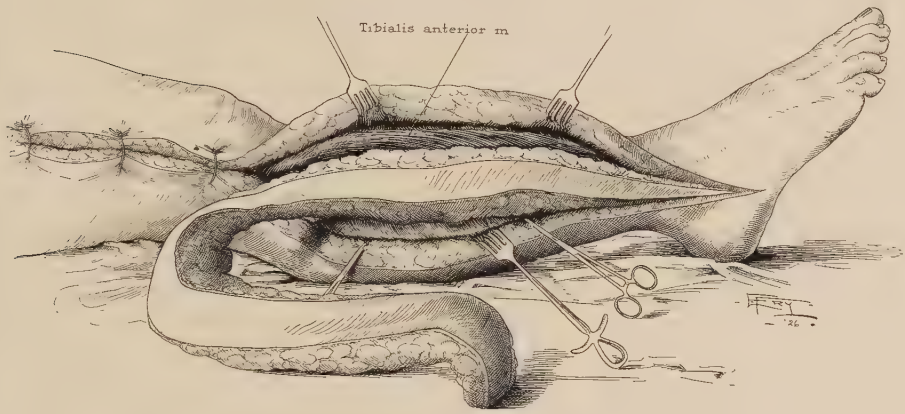

FIG. 10.—Incision in thigh closed and strip ready to be removed.

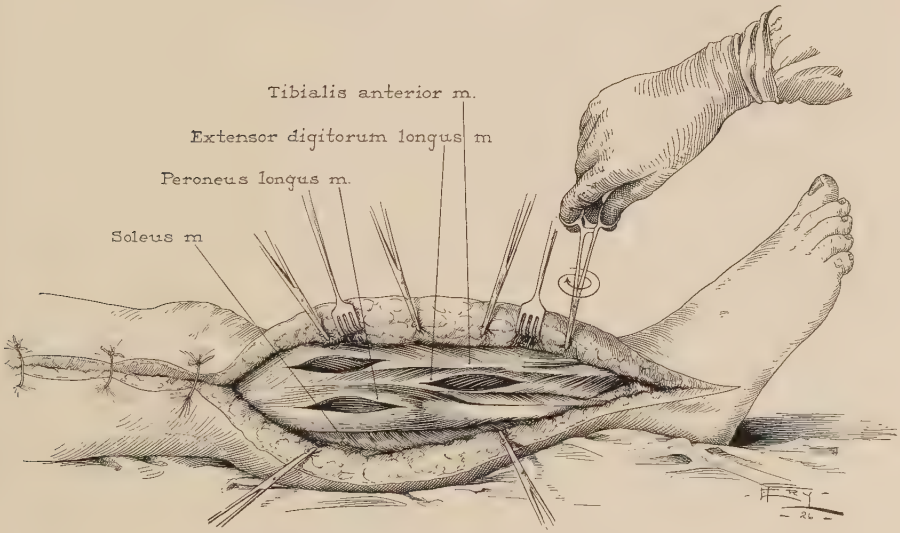

FIG. 11.—Incision in thigh closed, strip removed from leg and slits in muscles.

tion of opiates, usually $\frac{1}{4}$ grain of morphine, one-half hour before the operation and by deep ether anesthesia. Nerve-blocking, no doubt, would also help to prevent shock, although I have never

used this method. After the operation the patient is immediately treated as though severe shock were present—the head of the bed is lowered, external heat is applied, morphine is administered and isotonic solution of sodium chloride is given subcutaneously and by rectum. From 5 to 10 minims of a 1 to 1000 solution of epinephrin is also administered shortly after the operation, and repeated each hour for several doses. The period during which shock develops is usually passed after from six to twelve hours.

It is best before the operation to have a distinct understanding with patients as to the result to be expected; they should clearly understand that the operation is an attempt to control a disease which grows progressively worse if left untreated and which will eventually produce tremendous deformity and often in the late stages of the disease result in death through repeated attacks of erysipelas. They should also understand that it will be necessary to use a bandage for an indefinite period after the operation. Should extensive edema develop after operation in spite of the bandaging, much benefit is received by an occasional rest in bed with the limb elevated and carefully bandaged.

FREE, FULL-THICKNESS SKIN GRAFTS

Method of Making and their General Applicability, Particularly in the Treatment of Rodent Ulcers

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AND

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Ollier-Thiersch skin grafts are very simple in the technic of making and most successful in their results. It is a procedure that most house surgeons carry out with great success. But Thiersch grafts have this disadvantage, they frequently ulcerate, and also contract, from the thinness of the epithelial covering, allowing shrinkage of the underlying connective tissue. Were the principles of the application of free, full-thickness grafts more generally understood, these grafts would replace Thiersch grafts in a large per cent of instances, and the successes with them would be just as great as with Thiersch grafts. The histological basis of the nourishment of these full-thickness grafts has been put on a firm scientific basis by Staige Davis, who has experimentally proved that for twenty-four hours nourishment is by imbibition of plasma from the host into the graft, followed, after twenty-four hours, by an actual anastomosis of life-sized capillaries between host and graft; and then takes place an upward growth of capillaries from the host inside the old vessels of the graft. The circulation is finally completed, not before the eighth day at the earliest, by the growth of new arterioles into the graft, when the blood supply becomes adequate to fully nourish the graft. He has also shown that by removing the fat from the graft by scissors, capillaries are pinched and become occluded, consequently, it is wiser to remove the fat by a sharp scalpel. Only by careful

FIG. 1.—Upper: Chronic *x*-ray dermatitis (sweating) with dime-sized squamous cancer ulceration in one palm. Removal of entire palmar skin of both hands down to palmar fascia. Lower; Full-thickness, free graft to left and pedicled abdominal flap to right. Notice (right) redundancy of pedicled abdominal flap, due to fat; a disadvantage, requiring secondary removal of fat to make a good cosmetic result.

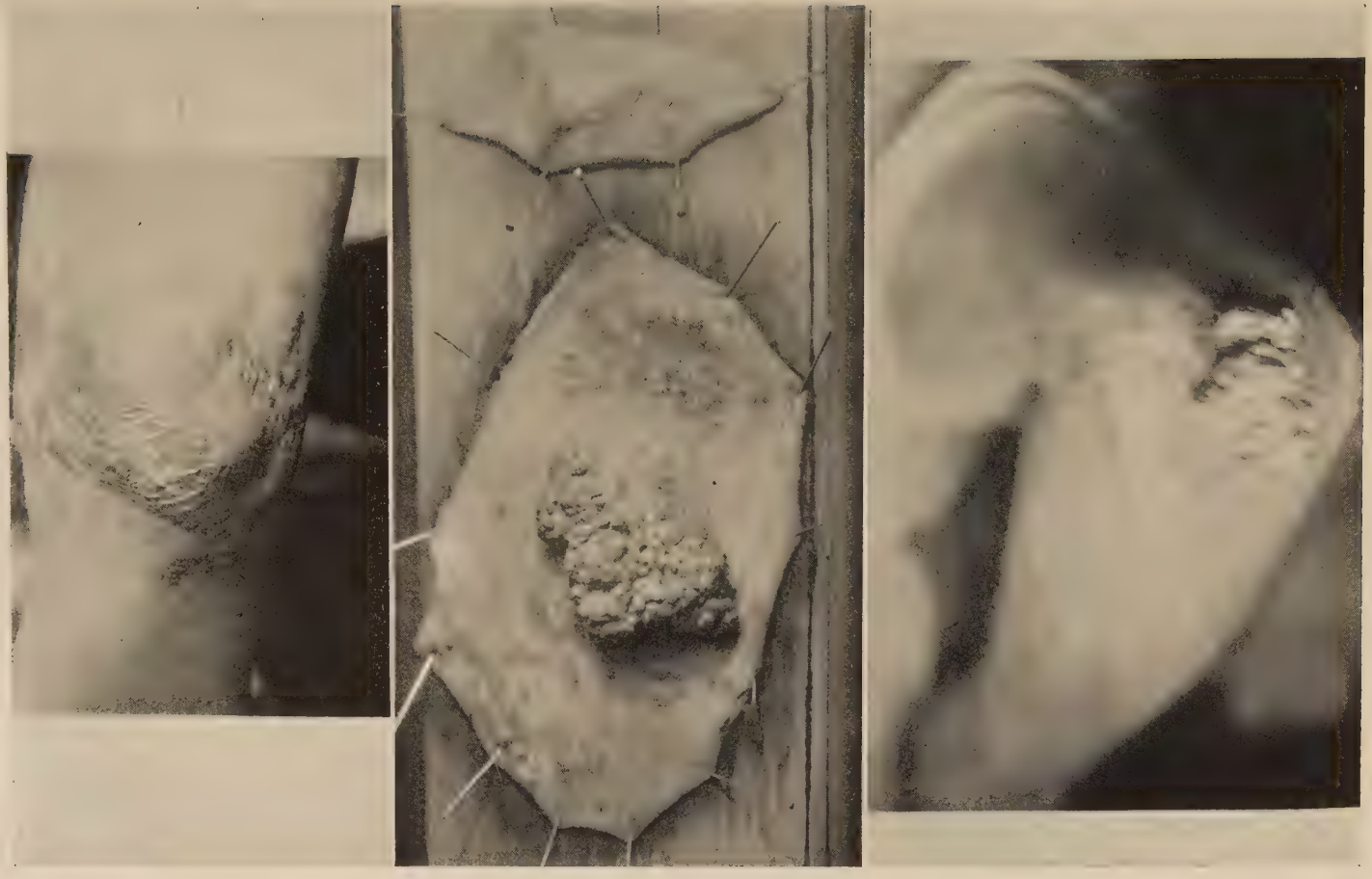

FIG. 2.—Left: Tuberculosis verrucosa cutis of elbow. Middle: Upper area excised, including tumor and affected skin. Right: Result from full-thickness graft, with perfect flexion and extension of forearm. Severe test of full-thickness graft.

attention to minor details is one sure of success with full-thickness grafts. Ferris Smith has computed the previously unknown amount of pressure to be applied to these grafts, to insure success, by ascertaining, by experiments, that 30 mm. of pressure is just the proper degree. He has devised (Fig. 8) an inflatable, rubber balloon to be incorporated in the dressing over the graft. This is a great advance in technic and takes the procedure out of the realm of uncertainty, for with the ordinary varying amounts of pressure obtained by a sponge, bandaged over the grafts, one never knows just the degree of pressure that is obtained. Probably this changing and uncertain amount of pressure explains the non-success of this method of grafting in the hands of many surgeons, for just the right degree of pressure is essential—for pressure conditions the nourishment of the graft; too much pressure will close the capillaries, shutting off the blood supply, and too little will allow the floating off of the graft from its underlying raw base by the effused serum, each being fatal to the life of the graft; hence, the great advance Ferris Smith has made in ascertaining just the amount of pressure necessary to insure success, and also an accurate method of applying that degree of pressure best, by means of an inflatable, rubber balloon.

The technic of making full-thickness grafts is more exacting than is the making of Thiersch grafts, but is soon mastered, and my house surgeons are now just as expert in fashioning them as Thiersch grafts. The method of technic is as follows:

1. Autographs are always to be chosen since isographs are very uncertain.

2. Fresh, sterile operating wounds, and fine, granulating (sterile) areas are equally successful.

3. Fresh fat and bone, bare of periosteum, are not usually successful areas to be grafted upon with free, full-thickness grafts.

4. Where contractions should be avoided (as about eyes and joints), free, full-thickness grafts should always be used rather than Thiersch grafts.

5. Grafts must be free of fat, which should preferably be removed by knife rather than scissors, to avoid pinching the capillaries.

6. No pinching of grafts with forceps is allowable.

7. The grafts are to be cut of exactly the same size (no larger) than the raw area to be grafted so as to preserve the normal tension of the skin to keep the capillaries open.

8. Making perforations in the graft is not essential.

9. The grafts should be sewn in all about the edges accurately with close, interrupted stitches, so as to maintain the normal tension, thus keeping the capillaries open.

FIG. 3.—Squamous-celled ulceration back of hand, duration five months, size of dollar. Excision, exposing tendon sheaths. Severe test of full-thickness graft, owing to poor circulation in base. Excellent result without any stiffness of fingers. Axillary nodes dissected and found uninvolved. Well one year after operation.

10. Even, firm pressure of 30 mm. should be applied upon the grafts by means of an inflatable, rubber balloon (Ferris Smith).

11. Absolute immobilization is essential for at least eight days so as not to disturb the growing capillaries.

12. There is no limit to the size of the grafts which may be successfully transplanted.

Successful Situations for Full-thickness, Free Grafts. 1. When applied on fresh muscle and fascia, including pericranium and periosteum. (2) On bared sheaths of tendons, whether granulated or fresh (Fig. 3). (3) On palmer fascia of hand (Fig. 1). (4) On fine, clean, granulating areas generally,.

Unsuccessful Situations. (1) On bone, bare of periosteum or pericranium. Bare skull bones are successfully grafted with

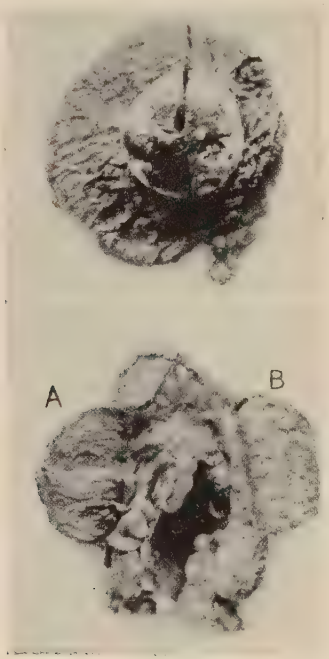

FIG. 4.—Squamous-cell epithelioma, removed from forearm, with wide excision locally, and dissection of axilla (uninvolved). *A B*, tumor cut open.

Thiersch grafts. There are not sufficient capillaries on fresh, bare skull bones to nourish full-thickness grafts. (2) On fresh fat. This should be allowed to granulate first if grafting with full-thickness grafts is contemplated. (3) Around the larynx, movements are constant from coughing and swallowing, despite all plaster-of-Paris devices. Ferris Smith has suggested wiring the teeth together to prevent movement through the lower jaw. (4) The face is an admirable situation for full-thickness, free grafts. (5)

Over joints Thiersch grafts should not be used because of their tendency to contract. Free, full-thickness grafts, or pedicled flaps, should be here employed.

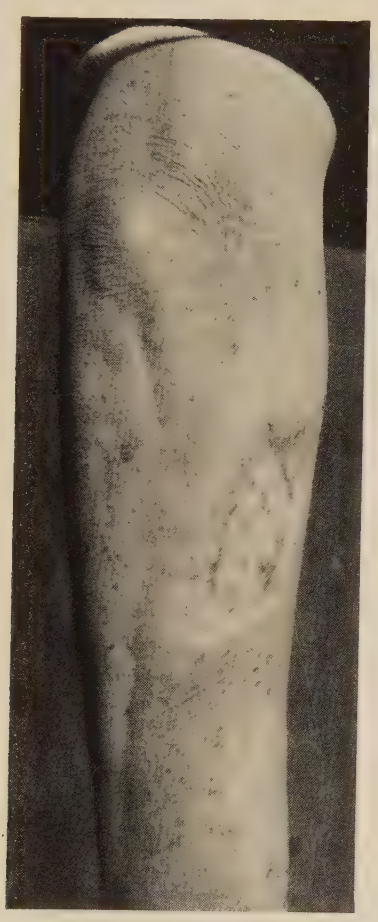

FIG. 4*a*.—Result of grafting free, full-thickness skin graft into defect of Fig. 4. Well three years later.

One of the great advantages of the full-thickness graft is its frequent mobility on the deeper parts (Fig. 2). This seems to be due to the fact that the fine connective-tissue layers underlying the normal skin are not entirely removed with the fat. These strands

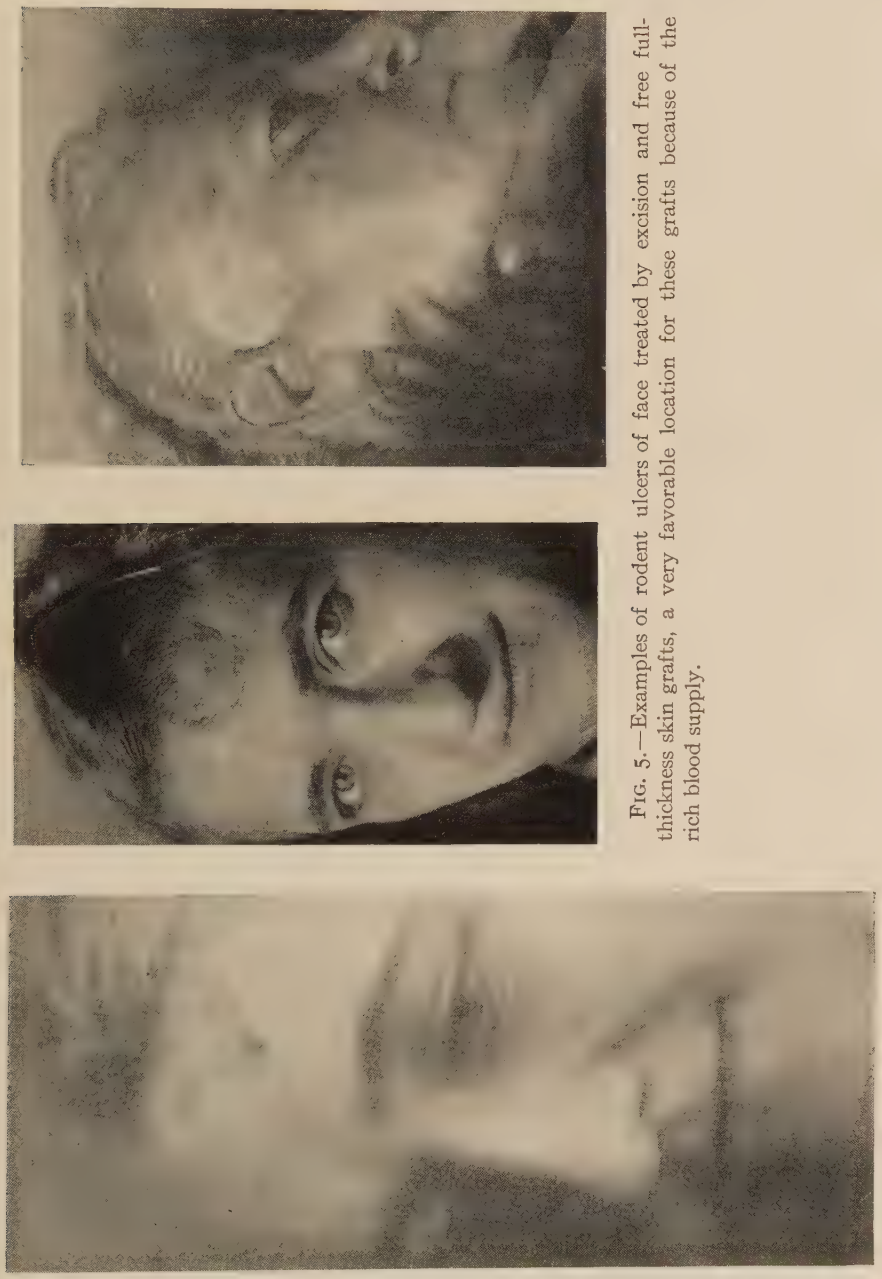

FIG. 5.—Examples of rodent ulcers of face treated by excision and free full-thickness skin grafts, a very favorable location for these grafts because of the rich blood supply.

seem to proliferate, affording movement to the skin graft overlying them. It has been observed that, at the first dressing, there frequently result several necrotic areas on the surface of the grafts, and the appearance is very discouraging for final success. But much to one's astonishment, these areas usually fill in with healthy skin without scar formation, due to the fact that the *entire thickness* of the graft has not necrosed away, and there is epithelial regeneration completely from the depths as well as from the sides, just as happens when the outer surface only of the skin is excoriated away. This heals usually without a scar. When a wound goes through the entire thickness of the skin, a scar will usually result because of the separation of the edges and the filling up of the cavity by connective tissue from that lining the bottom.

An ideal application of free, full-thickness grafts is in the treatment of the defects resulting from the surgical removal of rodent ulcers (Fig. 5), provided that the whole thickness of the cheek, opening into the mouth, has not been excised, in which case pedicled grafts will be required. Whether radium should first be tried in the treatment of rodent ulcers, rather than surgical excision, the author, as the result of experience, has for his own guidance formulated the following rules: If the ulcer be not large (*i. e.*, over 4 cm. in diameter) or of not too long duration (over one year), and if it does not involve bone or cartilage, then the results are equally as good with radium as with surgical excision, and the choice as to the variety of treatment may safely be left to the patient. When the above conditions are not present, however, surgery had best be used in the beginning. The technic of a full-thickness graft is a much simpler method than a pedicled graft, a method which has been so ably presented before this Association by Doctor Horsley. There is only one operative procedure necessary in making full-thickness grafts, and there is no additional scarring. It can be frequently done under local anesthesia in one's office. It would seem to the author that a free, full-thickness graft should first be tried after excising the ulcer, and, if it be not successful, one always has then, as a last resort, the successful pedicled graft. If the technic of making full-thickness grafts is carefully carried out as to details, and is not modified, the success thereby obtained will be just as frequent as with Thiersch grafts. My own personal experience with full-thickness grafts applied to granulating areas has been small, but

regarding this, Staige Davis, in a personal communication, says: "My first use of the whole thickness graft was on granulating surfaces, and I find that, if the granulations are flat and clean, that whole-thickness grafts take just as well, if not better, on granulating surfaces than on fresh ones. I account for this by the fact that the granulating buds have already started and penetrate the grafts rapidly."

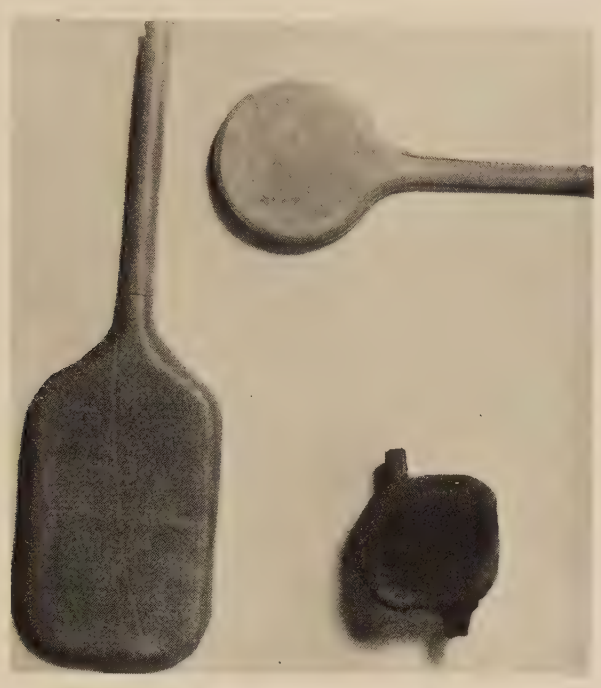

FIG. 6.—Various forms of Ferris Smith's inflatable rubber balloons. To be incorporated over full-thickness grafts in the dressing, and inflated to 30 mm., exercising just the proper degree of pressure (important point in technic). Lower right, two-way inflatable, rubber balloon over nose, so that it can also be used with continuous flow of cold water for inflammations.

With the additional pressure balloons of Smith (Fig. 6), we have a procedure in full-thickness grafting which is precisely scientific, exact in all its details, accurately worked out, and should give surgeons 95 per cent of favorable results, with as great a frequency of successes as after using Thiersch graftings. In my own practice full-thickness grafts are now employed twice as frequently as Thiersch grafts.

A Discussion of the Treatment of Rodent Ulcers by Radiation

By RAYMOND W. LEWIS, M.D.

Radiation treatment of basal-cell epitheliomata is popular with dermatologists and radiologists because their common experience is to observe a rapid disappearance of most of these lesions under this form of treatment, with excellent cosmetic results. The method is further liked by both physician and patient because of absence of operative procedure and for its simplicity of application.

Those who oppose this form of treatment do so in the belief that the uncertainty of a permanent cure is very great, and that there is a large percentage of recurrence. This belief does not find support in the figures of careful observers. Hazen[1] recently reported a series of 244 basal-cell tumors treated by radiation, with 33 failures or recurrences from the first series of irradiation (about 14 per cent); and 5 of these were cured by a second course. MacKee[2] followed 282 clinically cured patients for periods varying from six months to nine years, and found 36 relapses, 13 per cent. As a basis for comparison, Hazen[3] collected a series of surgical results from Johns Hopkins Hospital and found 86 per cent of cures in unselected patients. The percentage of cures from the two methods would therefore seem equal, and both fairly satisfactory.

A second, and better founded cause for opposition to radiation treatment, is found in the occasional patient who has had an extensive or resistant lesion, which has been treated over a long period of time, with many applications of *x*-ray or radium, and who eventually comes to surgical treatment with such an extensive lesion, and with tissues so badly damaged by radiation, that he presents to the surgeon a well-nigh hopeless condition.

Such unfortunate results, and grounds for opposition to radiation, could be easily avoided if we would frankly recognize at the start the limitations of this method of treatment, and not attempt too much. If there is not an immediate response to one or two doses of radium, surgical excision should be resorted to. Rodent ulcers involving bone or cartilage are cured with the greatest

difficulty, if at all, by radiation. And very large (roughly, 4 cm. in diameter, or over) or very old lesions are difficult to cure. It is our feeling that most lesions of these types should be seen in consultation by a surgeon experienced in cancer work, and treatment of them by radiation should be undertaken only if the surgeon considers they are for any reason unsuitable for operation.

The third objection to radiation is based on the occasional error in diagnosis which results in the treatment by radiation of a supposed basal-cell growth, which eventually proves to be squamous celled. Whether the treatment of squamous-cell epitheliomata by radiation should be undertaken or not, we do not propose to discuss in this paper, but the fact is well recognized that this latter form of growth is very much more difficult to cure by *x*-rays and radium than the former. But we do not feel that such a mistaken diagnosis adds very much risk to the patient if the following rule is adopted: Whenever a lesion shows any increase in size after treatment, or whenever a lesion does not show very striking response to one or two doses of radiation, the patient should be referred at once for surgical removal and pathological examination of the specimen. If this rule is followed, the patient reaches the surgeon with the lesion still small, and with little radiation damage to the surrounding tissues, and the dangers of recurrence have probably been increased little, if any. We feel, also, that this rule is equally desirable for the occasionally encountered very resistant basal-cell tumors.

Our figures from the New York Skin and Cancer Hospital are given as a preliminary report only, owing to the recent organization of its present department of roentgenology. Only the radium treated patients are used in the following figures as our records of these patients are more complete.

Our records show that during the past twenty-two months we have carried through to completion the radium treatment of 50 patients with clinically diagnosed basal-cell epitheliomata. Among these there were five primary failures, or 10 per cent. One of these, which involved an extensive area of one cheek, we had from the start considered too large to be suitable for radiation; and although the lesion showed considerable improvement from two applications of radium, we persuaded the patient to consult one of our surgeons, feeling that surgery offered the better prospect of permanent cure.

FIG. 7.—Results of radiation on rodent ulcers. *A*. Three applications, the last one twelve months prior to the photograph. *B*. Two applications, the last one seven months prior to the photograph; permanent epilation of a few lashes of the lower lid. *C*. Two applications, the last one six months before photograph.

The pathological report of 2 of the remaining 4 failures, surgically removed, was squamous-cell epithelioma. An interesting feature was that 1 of these patients gave a history of three years' duration of the lesion. Both these patients have been seen within a month, and are well, three months and ten months after excision.

The remaining 2 of the 5 failures were also referred to the surgical department and operation advised, but did not come into the hospital. If we assume that their lesions were basal-cell epitheliomata, we have in our series about 4 per cent of these lesions found entirely resistent to the usual dosage of radium.

We have been able to follow 29 of our clinically cured patients for periods varying from three to nineteen months after their last radium application, a general average of seven months. There have been 3 recurrences in this series, about 10 per cent. One of these recurred seven months after her last application of radium. She received another treatment, and is well at present, seven months later. A second patient had a recurrence five months after treatment. She received another application of radium, but did not report in again, and we have been unsuccessful in our attempts to follow her up. The third patient had a recurrence in one of a number of lesions we treated on his face, four months after completion of treatment. He has received another radium application on this lesion within the past few days.

SUMMARY. 1. Radiation treatment of basal-cell epitheliomata is an excellent method because of its absence of operative procedure, its simplicity, excellent cosmetic results, and high percentage of permanent cures.

2. Statistically, there is little difference in the ultimate results between treatment by radium and that by surgical excision.

3. The treatment by radiation of those lesions where bone or cartilage is involved, or where the lesion is 4 cm. in diameter, or larger, is inadvisable. Treatment by radium should be undertaken in these conditions only when a surgeon, skilled in the treatment of cancer, finds the patient, for any reason, unsuitable for operation.

4. The patient's risk, in treating, through mistaken diagnosis, a squamous epithelioma, is probably little, if any, increased, if the rule be observed to refer promptly for surgical removal any lesion which increases in size after radiation, or any lesion which

does not respond immediately and strikingly to one or two doses of radiation.

5. A preliminary report of basal-cell lesions treated by radium at the New York Skin and Cancer Hospital is given. The authors desire to express their thanks to Doctor Watters, of the Interne Staff of the New York Skin and Cancer Hospital, for the excellent photographs.

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A PLASTIC OPERATION ON THE CHEST WALL

By ARTHUR M. SHIPLEY, M.D.
BALTIMORE, MD.

I AM reporting this case to call attention again to the use of wire and buttons in plastic surgery, and because it is further evidence that the clean chest can ordinarily be opened and closed without special anxiety and without elaborate preparation as to the anesthetic.

I saw this patient for the first time in April, 1919. She had a lump in the left breast which had been present for three and a half years. She had been under treatment at irregular intervals by her physician, who had given her potassium iodide.

The mass was not large and occupied the central portion of the breast. There was considerable retraction of the nipple, but there had been no ulceration of the skin about the nipple previous to the appearance of the lump. The patient was very thin, but this was not a recent development as she had always been thin and frail in physique. The breasts were both small, with very little muscle between the breast and ribs. The mass in the left breast was adherent to the chest wall and to the skin and there were a number of hard, small, shotlike masses in the left axilla. The patient's general health was not impaired.

A radical breast amputation was done and great care taken to do a complete axillary dissection. The pathologist's report showed scirrhous carcinoma of the breast with metastasis to the axillary lymph nodes. The patient made an uneventful recovery and the skin united everywhere.

In March, 1923, four years later, she came to see me again, and at this time there was a small ulcer in the scar. It had been there for six weeks. This ulcer was small and no other evidence of recurrence could be made out, either in the skin of the chest wall, in the axilla or in the neck. This small ulcer was excised under local anesthesia and it was reported scirrhous carcinoma.

In April, 1929, I saw her again. It was now six years since the first

operation and two years since the small ulcer was removed. At this time there was extensive recurrence of the skin and chest wall over the site of the amputated breast. There was no evidence of recurrence in the axilla or metastasis in the neck and the roentgen-ray examination

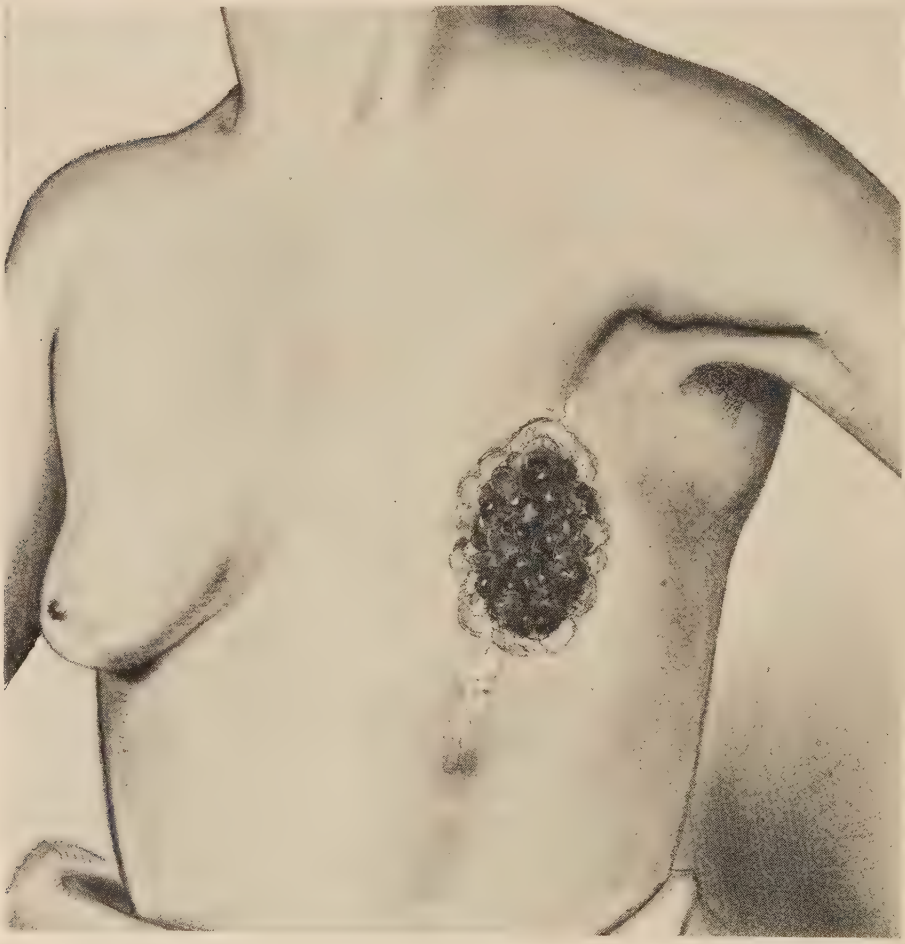

FIG. 1.—Recurrent carcinoma of breast.

of the chest was negative. The patient's general health was good, although she was still very thin. Because of the extreme thinness of the chest wall and the extent of the malignancy, it was believed that it might be impossible to get away the carcinomatous mass without

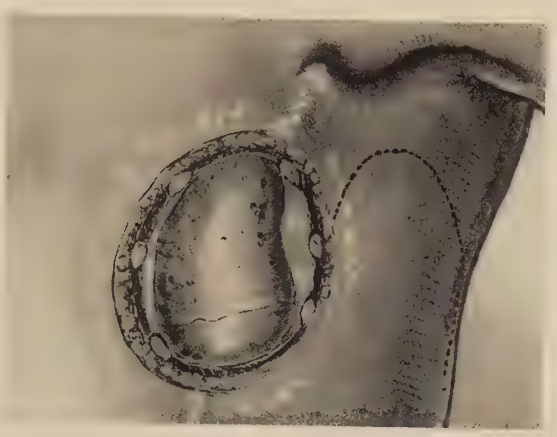

FIG. 2.—Excision of a portion of the chest wall.

FIG. 3.—Defect in the chest wall showing different structures.

opening the pleural cavity, so that preparation was made for differential pressure anesthesia. It was hoped, however, that it would be possible to separate the chest wall from the pleura and that the pleura would be found free from cancer. There was no effusion in the pleural cavity. The cancer had infiltrated the entire thickness of the chest wall, how-

FIG. 4.—Flap of skin, fascia and muscle used to cover defect.

ever, and as this was the second local recurrence without general metastasis, it seemed useless to do anything less than a radical excision of the mass. This would be difficult, because the previous operation had removed all muscle and fascia from the ribs. The skin was firmly adherent to the ribs and there was considerable involvement of the bone and periosteum.

An attempt was made to separate the mass from the underlying pleura, but without success, so that in getting away the carcinomatous area a large defect was made in the chest wall, including the pleura. The lung collapsed on this side to a moderate degree. The breathing of the patient was not disturbed to any considerable extent and the

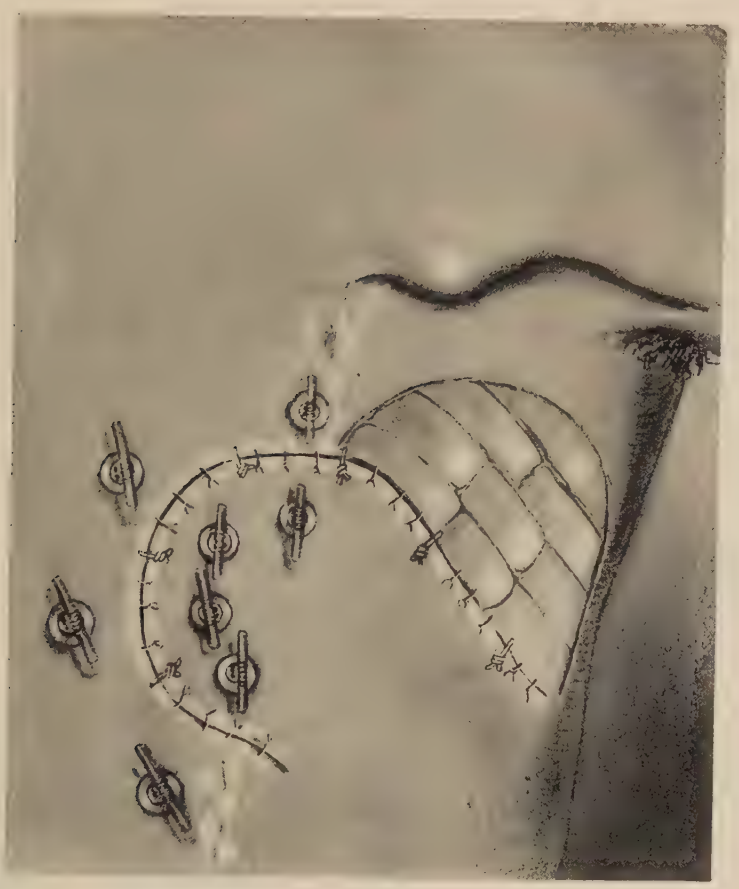

FIG. 5.—Completed operation, Thiersch grafts used to cover denuded area.

heart action continued about as it was before the chest was opened. There was no mediastinal flutter. The opening in the chest wall, however, was kept closed with gauze, as much as possible, during the entire time that the thorax was open.

Closure of the defect presented a difficult problem, because of the extensive previous operation which had removed all muscle and fascia

in the immediate neighborhood, and because, as mentioned before, of the patient's extreme thinness. Sliding the opposite breast across the chest on the defect was considered, but this was not possible, as the opposite breast was very small, there was no subcutaneous fat and very little muscle and the skin everywhere was quite taut.

FIG. 6.—Photograph of patient one year after operation.

A pedicle flap was fashioned, therefore, from the axilla on that side, with the base down. This flap was taken from over the serratus magnus muscle and lateral to the original field of operation. It was still a very thin flap and considerable doubt was felt as to whether it would live.

A broad base was left, however, and it was turned into the defect with very little tension. The edges of the original wound had been undermined about 1 inch, and tension was removed from the suture line by the use of silver wire, buttons and pieces of matchstick. The wire was carried through the skin on a heavy needle and brought out on the skin about 1 inch away from the wound edges. The buttons were threaded down on the end of the wire and the wire tightened by wrapping it around pieces of matchstick. This is a very simple maneuver and the sutures can be tightened or loosened at will, by twisting or untwisting the matchstick. The edges of the skin were then approximated by interrupted sutures of fine silk. These were interspersed at intervals with interrupted sutures of wire. Care was taken to make the suture line air-tight.

The defect in the lateral chest wall was covered by Thiersch grafts taken from the thigh. During this time the breathing remained quiet, the heart action regular and the color of the patient good. The lung continued in a moderately collapsed condition, but there was very little movement in the lung on that side during respiration and no gross interchange of air through the wound during respiration. No attempt was made to expand this lung before putting in the last suture. An ordinary dry dressing was applied and the chest wall strapped moderately snug.

The patient made an uneventful recovery. The pneumothorax rapidly disappeared, no effusion occurred in the chest and the wound healed promptly throughout its extent and when seen several weeks ago was in good condition, without any sign of recurrence.

OPERATIVE TREATMENT OF THENAR PARALYSIS

By HENRY H. M. LYLE, M.D.
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The purpose of this paper is to call attention to certain operations which are valuable in the treatment of thenar paralysis. We assume that nerve suture has been tried or that it is impracticable.

One of the difficult problems of reconstructive surgery has been to devise a suitable tendon transplantation to replace the intrinsic thumb muscles. The functional loss of these muscles makes the thumb useless and destroys over 75 per cent of the efficiency of the hand. In thenar paralysis the thumb falls back into the same plane as the other fingers and cannot be opposed.

Stiles, in 1922, stated that no satisfactory tendon transplant had been devised to replace the intrinsic thumb muscles and advised an arthrodesis of the first carpometacarpal joint. Platt and Bristow (1924) state that tendon transplants are occasionally of service in assisting function, *e. g.*, after lesions of the median nerve to restore some power in the opposing thumb, but these are not commonly practised.

The type of operative procedure to be employed depends on the presence or absence of suitable functionating tendons. If these be present, some form of tenoplasty can be used; if absent, arthrodesis or fascial anchorage will be necessary.

The pioneer in this work has been Steindler, of Iowa; the other contributors are Cook, Ney and Bunnell. In Germany, Kortzeborn has devised a combined fascial transplant and plastic for "ape hand." In 1918 Steindler described his operation for the plastic substitution of the opponens action of the thumb. It is a flexor plasty of the long flexor of the thumb (Fig. 2). This operation yields an excellent functional result. Cook's operation was described by R. Taylor in 1921. It is a transplantation of

the extensor minimi digiti to the distal end of the first metacarpal. The tendon is passed to the palmar surface and then through the subcutaneous tissue to be fixed into the first meta-

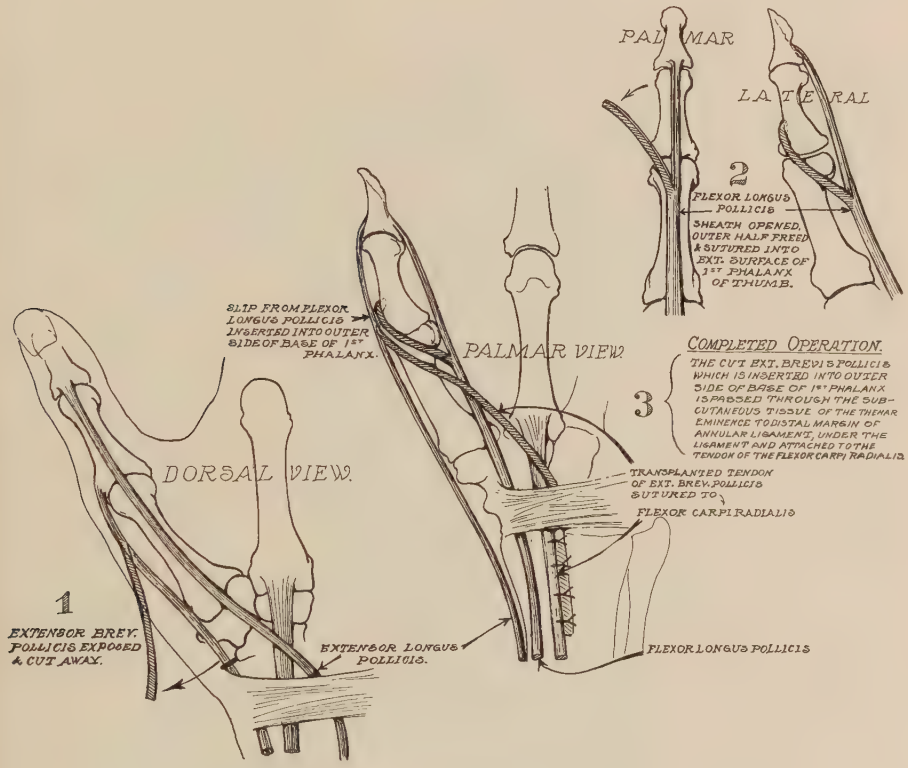

FIG. 1 FIG. 2 FIG. 3

FIG. 1.—Extensor brevis pollicis exposed and cut away.

FIG. 2.—Flexor longus pollicis sheath opened; outer half freed and sutured into external surface of first phalanx of thumb.

FIG. 3.—Completed operation. The cut extensor brevis pollicis, which is inserted into outer side of base of first phalanx, is passed through the subcutaneous tissue of the thenar eminence to the distal margin of the annular ligament, under the ligament and attached to the tendon of the flexor carpi radialis.

carpal. Cook has operated on 4 cases; in the 3 cases that he has been able to follow the results were very satisfactory.

Ney published his operation in 1921. It consists of a trans-

plantation of the extensor brevis pollicis into the palmaris longus, or in the absence of this muscle to the flexor carpi radialis. The operation gives a satisfactory functionating thumb (Fig. 1).

FIG. 4.—Shows the split tendon of the flexor longus pollicis, the outer limb of which has been carried subcutaneously around the outer side of the base of the first phalanx and fixed to it. The tendon of the extensor brevis pollicis has been carried subcutaneously across the thenar eminence, passed under the anterior annular ligament and sutured to the tendon of the flexor carpi radialis.

Bunnell, in 1924, in a case of infantile paralysis involving the extensors of the fingers and the intrinsic muscles of the thumb transplanted the palmaris longus into the extensor longus pollicis. The tendon of the palmaris was passed through a pulley at the

pisiform bone and then through the subcutaneous tissue to the long extensor tendon of the thumb. The pulley was made out of a free tendon graft obtained from the extensor tendon of the toe. This gives an oblique pull, somewhat similar to Cook's operation and, like it, a good power of opposition.

FIG. 5.—Before operation. Paralysis of the intrinsic muscles of the hand; duration, thirteen years. Note the thenar and hypothenar atrophy, absence of thenar crease, flat palm and the position of the thumb, which lies in the same plane as the other fingers. Compare with normal hand.

In those cases where the thumb assumes the flexed position when opposed to the fingers, from lack of power of extension, the tendon which is to give the opposition to the thumb can be attached to the tendon of the extensor longus pollicis at

the metacarpophalangeal joint. The thumb will then properly oppose the finger.

The writer has employed both Steindler's and Ney's operations, and obtained very satisfactory results.

FIG. 6.—Result after operation. Note the power of adduction and presence of the thenar crease. Patient now follows his trade.

The resulting pincer action of the thumb, though strong enough for ordinary use, is not quite powerful enough in some cases. In an effort to improve this essential action, we have tried to combine the advantage of Steindler's flexor plasty with the good points of Ney's operation. The operation consists of two steps: (1) The extensor-flexor plasty (Ney) and (2) the flexor plasty (Steindler) (Figs. 3, 4, 5, 6, 7 and 8).

1. An incision is made over the extensor brevis pollicis extending from its insertion to a point where this tendon emerges from the posterior annular ligament; the tendon of the extensor brevis pollicis is then divided at this level. An anterior incision exposing the flexor carpi radialis at the wrist is made, the subcutaneous tissue is tunneled obliquely from the distal edge of the anterior

FIG. 7.—Thenar paralysis of eight years' standing. Trauma to brachial plexus. Note thenar and hypothenar atrophy and the absence of the thenar crease. The palmar surface of the thumb does not face the palm directly but looks obliquely across. Any adduction is brought about by the flexor longus pollicis.

annular ligament to the insertion of the extensor brevis pollicis and the cut end of the extensor brevis pollicis threaded through this tunnel, passed under the annular ligament and sutured to the tendon of the flexor carpii radialis above the annular ligament. The palmaris longus, if present and sufficiently developed, can be employed instead of the flexor carpi radialis (Figs. 1 and 4).

2. Flexor plasty: A palmar lateral incision is made over the tendon sheath of the flexor longus pollicis, exposing it from its insertion to a point just below the head of the first metacarpal. The tendon sheath is opened and the tendon split longitudinally; the outer half is freed from its insertion and withdrawn from the sheath and the sheath is then closed over the internal half. The

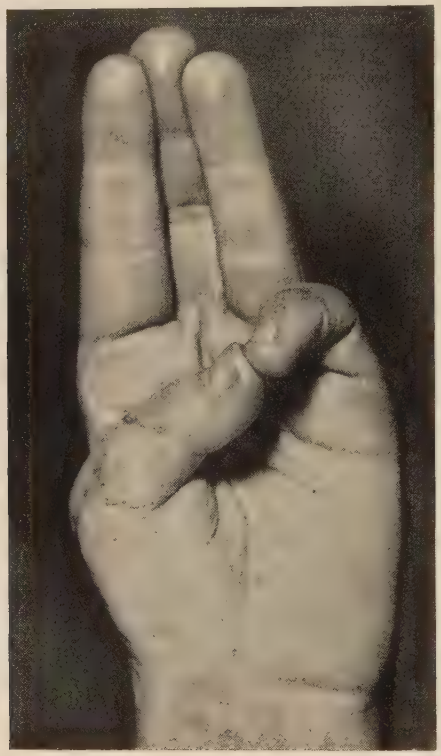

FIG. 8.—Result after operation. Note the adduction, the formation of the thenar creases and the restoration of the palmar hollow

external half is carried subcutaneously around the outer side of the base of the first phalanx and sutured. This can be readily done, as the previously performed incision for the exposure of the insertion of the extensor brevis pollicis gives an excellent exposure for the flexor anchorage. The thumb is then placed in the functionating position and fixed. Gentle active movement is begun

on the twelfth day; all support removed on the twenty-first day (Figs. 2 and 4).

The author reserves his operation for the cases in which it is essential to have a powerful pincer action. In the ordinary case he employs the simper methods of Steindler or Ney.

One of our patients, a skilled woodcarver and turner, sustained an injury thirteen years previously which left him with a thenar paralysis. This forced him to give up his trade, as he could neither grasp nor guide his tools. Six months after the above operation the patient resumed his trade and has followed it for two years.

FIG. 9.—Before operation. Paralysis of the intrinsic muscles of the left hand due to injury of brachial plexus. Note flat hand, thenar and hypothenar atrophy. Thumb cannot be adducted.

OPERATIVE PROCEDURES WHEN NO SUITABLE TENDONS ARE AVAILABLE. 1. Baldwin's operation for flat abducted thumb deformity is an arthrodesis at the basal thumb-joint to procure a short fibrous union in the functionating position. The pincer action of the thumb is now made possible, and this action is valuable in performing the delicate movements, but unfortunately the grip of the thumb is not restored. In 1922 Stiles advocated this operation, but stated that the operation only restored the pincer action for small, light objects. In our hands the functional results from this operation have been inferior to those of the tendon and fascial transplants.

2. Free fascial transplants: Where no tendons are available Bunnel has employed a free graft of fascia as a permanent check-

rein. The fascia is passed from the distal end of the first metacarpal to the pisiform in such a manner as to hold the thumb in

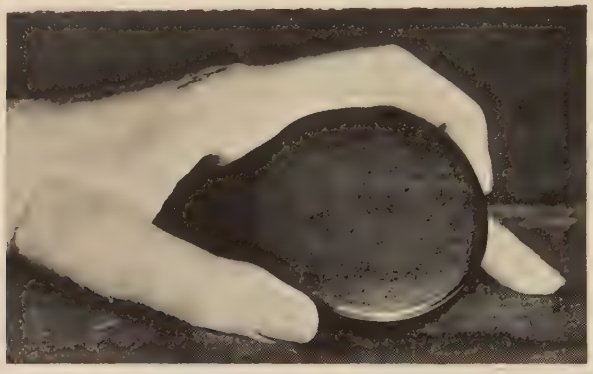

FIG. 10

FIG. 11

FIGS. 10 and 11.—After operation. Functional result eight weeks after an arthrodesis of the first metacarpophalangeal joint.

opposition. In 1924 Kortzeborn described his operation for "ape hand." He attempts to restore the hollow of the hand and fix the thumb in a functionating position of opposition.

The operation consists of a lengthening of the extensor tendons of the thumb, the fixation of the thumb in the position of opposition by a fascial transplant and a plastic on the palm.

Tenoplasty is indicated in thenar paralysis after nerve injury where nerve suture is impossible or has failed; as an aid to hasten functional recovery in selected cases of delayed nerve suture; in suitable cases of anterior poliomeylitis; in those cases of occupational thenar paralysis which fail to respond to treatment.

Care must be taken to exclude syringomyelia, intramedullary and intradural spinal-cord tumors, extravertebral and paravertebral tumors, cervical ribs, lesions and elbow-joint giving rise to nerve pressure, tumors of the nerves, leprosy, etc.

SUMMARY. 1. Thenar paralysis is a serious functional disability.

2. This disability has not received the therapeutic attention from the general surgeon that it deserves.

3. Suitable tenoplasties have been devised which yield a high percentage of functional improvement.

4. If tenoplasty is impossible fascial anchorage or an arthrodesis will improve this otherwise hopeless condition.

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ANATOMICAL STUDY OF COMPLETE UNILATERAL CLEFTS OF THE LIP AND PALATE

With a Description of the Method of Closure of the Defect in Early Life by Operations in Successive Stages

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One of the simplest and most rational classifications of congenital clefts of the lip and palate was suggested by Ritchie and Staige Davis in 1922. Taking the alveolar border as a fixed point, they divide the clefts into three groups, viz.: (1) prealveolar, (2) postalveolar and (3) alveolar clefts. In the first two the alveolar process is intact and the cleft affects the parts in front or behind it; whereas in the third the essential feature of the deformity is a faulty union of the component parts of the alveolar process.

Every cleft of the alveolar border is placed in the third group, whether the lip in front or the palate behind be open or closed. It is divided into three varieties as follows: (1) the *unilateral*, in which the cleft lies between the premaxilla and the maxilla of one or other side; (2) the *bilateral*, where clefts are present on each side between the contiguous margins of the premaxilla and maxillæ; and (3) the *median*, in which, owing to faulty development or absence of the premaxilla, the cleft lies between the anterior ends of the maxillæ.

It is my intention to base the description of the septal operation on the anatomical conditions found in the first of these varieties, viz., the unilateral, and to confine it to clefts implicating lip, alveolar border and the whole length of the palate, a type which I have been in the habit of calling a *complete unilateral cleft of the lip and palate.*

According to the classification of Ritchie and Davis, the formula of this deformity would read as follows:

ALVEOLAR CLEFT (GROUP III)

Alveolar process	Unilateral, complete
Lip	Unilateral, complete
Palate	Median, complete

As it is necessary for our purpose to make a careful study of the anatomical features of the deformity, we submit the following description, which is based on a careful study of all the cases observed during the last twenty years. As they usually conform to a common type, the illustrations which accompany the text have been taken from a few of the most typical examples.

ANATOMICAL DESCRIPTION OF THE DEFORMITY

In an average case the appearances are definite and clear-cut. The nostril flares wide open and the margins of the cleft in the lip are separated from one another by a considerable space, in which one can see the tips of the ununited alveolar processes of the maxillæ and the side of the anterior part of the septum. Through the open mouth one can see the cleft in the palate extending through the alveolar border in front and passing backward through the whole length of the palate to the tip of the uvula behind. The maxillæ are usually separated from each other by a considerable distance. In front, as measured between the tips of the alveolar process, they are often as much as 15 or 20 mm. apart; while behind, as measured between the tuberosities, the separation is seldom greater than 2 mm.

In order to understand clearly the reasons for the operative steps, which we shall describe later in detail, it is necessary first to describe every feature of deformity minutely.

THE NOSTRIL AND THE LIP. The appearance of the nostril and the lip depends on the degree of separation of the tips of the alveolar processes from each other. When they are close together the nostril may be fairly well formed and the margins of the cleft in the lip may lie fairly close together, but when they are wide apart the deformity is quite striking. In these cases (Fig. 1) we see a wide-open, flaring nostril bounded in front by the flattened ala nasi, whose natural curve is almost obliterated, except

at its outer end where it joins the cheek. The nostril on the sound side is usually well formed and free from distortion. If the nose and lip are viewed from the front the whole nose appears to slant to the side opposite the cleft, and if a pencil or ruler be placed along the bridge of the nose the degree of deviation can be measured accurately. It gives the impression that the nose,

FIG. 1.—Photograph of a case of complete unilateral cleft of the lip and palate. All the anatomical features, described in the text, referring to the nostrils and the lip on both sides of the fissure are very clearly shown. On the right margin of the cleft the curved line separating the surface of the lip from the lining of the nostril is particularly clear. On the left side of the cleft one can see very clearly the shearing of the philtrum, the boundary line between the philtrum and columella, and the triangular area just below the intact nostril which represents the lateral nasal process.

septum, premaxilla and the maxilla of this side (which we shall call the "greater process") have been pushed bodily forward and slightly outward, for a considerable distance. If a line be taken from the root of the nose to the middle of the mandible the exact extent of the deviation of the greater process from the middle line of the face can be demonstrated graphically. We shall point

out later that the opposite maxilla ("lesser process") has barely moved at all and lies almost in its normal position.

The lip on the inner margin of the cleft differs greatly in appearance and structure from that on the outer margin. On the inner margin (over the greater process) the lip is thin and scanty.

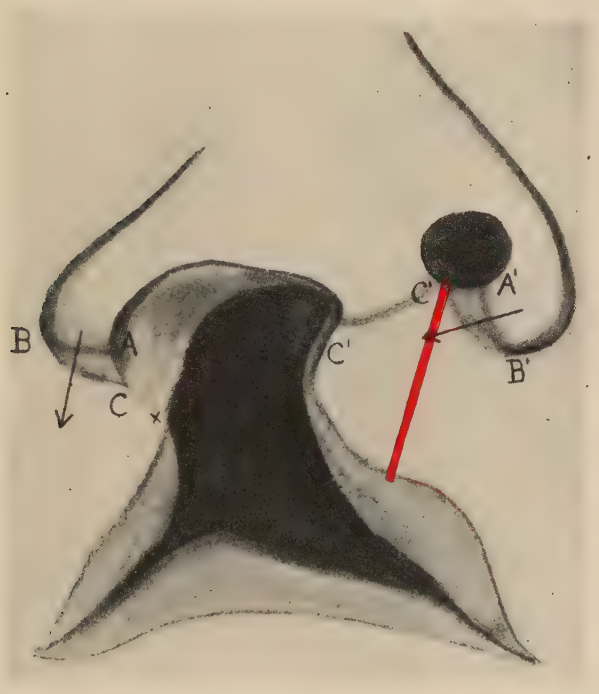


FIG. 2.—An enlarged projection picture drawn from a lantern slide made from the photograph of the case in Fig. 1. The flaring nostril has been drawn so as to bring in relief every detail shown in the photograph. The arrows on either side show the direction in which the ala nasi points. On the side of the cleft the line of demarcation between skin and nasal mucous membrane is shown along the curved line $\times$ *C A C'*. *The red straight line* shows the measurement taken to determine the depth of the future lip. Other detailed description is given in the text. The curved shaded line between *C'* and *C'* shows the boundary between the philtrum and columella.

Its free border often shears off so abruptly to the corresponding angle of the mouth that the lower portion of the philtrum is frequently missing. Its vermilion border is usually well developed and reaches upward as far as the inner margin of the nostril. The deep surface of the lip is firmly attached to the underlying

bone and a well-developed frenum is invariably present. Owing to the unbalanced action of the facial muscles on this side, the lip is frequently pulled so far away from the cleft that the tip of the alveolar margin of the greater process is completely exposed. The lip on the outer margin of the cleft (over the lesser process)

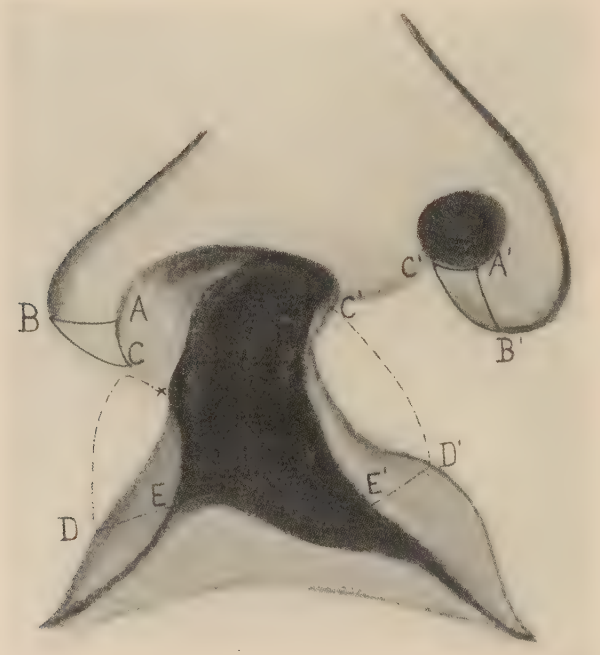


FIG. 3.—The same picture as Fig. 2. The interrupted lines (dash, dot) show the usual method of planning the incisions in order to secure accurate coaptation of the sides. This method will restore the curve of the nostril, make the lip of the proper depth and restore the vermilion border. It will not, however, bring the ala nasi into its correct position. In this picture and in Fig. 5 the upper end of the incision *D C* should not cut its way into the margin of the cleft by passing through the nasal mucous membrane. It should follow the curved line × *C*. By this means all the nasal mucous membrane is preserved.

is usually thick and well formed. It covers completely the maxillary process on which it rests. Its free border lies more or less vertical and it joins the oral margin of the lip almost at a right angle. The vermilion border is usually well developed. It can often be traced almost as far upward as the margin of the nostril.

If on each side of the cleft we follow the free edge of the lip into the nostril we shall find the following appearances: On the inner side of the cleft (over the greater process) the free border of the lip becomes less prominent as we pass upward until we reach a

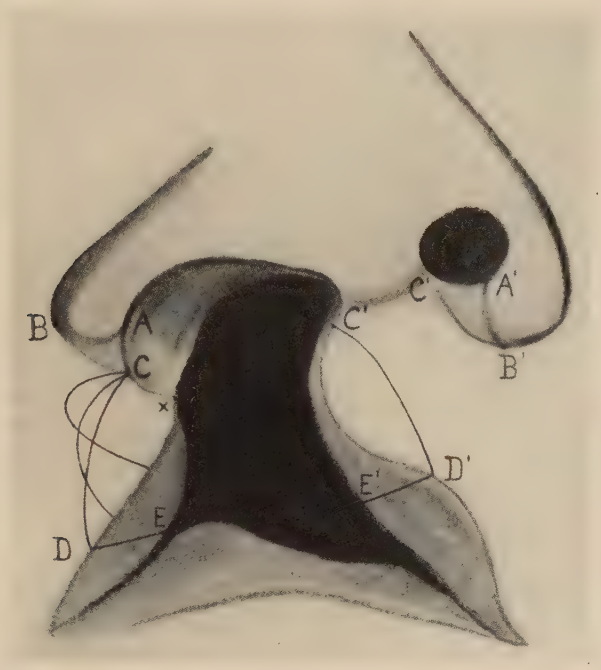


FIG. 4.—On the left of the picture a slightly curved line, *C D*, is shown, which is identical with that in Fig. 3. In addition, two other curved lines are shown stretching from the point *C* to the vermilion border of the lip, along the line *x D*. Each of these lines is of equal length and each is equal to *C D*. The upper line has the most pronounced curve, and its upper end bends somewhat abruptly underneath the outer horn of the ala nasi. Lines through the mucous membrane of the lip, corresponding to *D E*, are purposely omitted to avoid crowding the diagram. The figure shows the necessity of carrying the lateral incision *C D* outward under the point *B*, in order to bring the line *A B* in a position similar to *A′ B′*, when the lines *C D* and *C′ D′* are approximated. We frequently use the line with the greatest curve.

point where the columella and philtrum meet, where it blends with the mucous membrane forming the medial wall of the nasal passage. This point is usually shown on the surface of the philtrum by a groove, more or less obliquely placed, which separates the columella from the philtrum. Where the groove meets the

margin of the sound nostril it indicates the point of union of the apex of the lateral nasal process with the side of the medial nasal process; where it meets the margin of the incompleted nostril it indicates the place where the two nasal processes ought to fuse together under normal conditions and shows accurately the height to which this side of the cleft can be denuded when paring it for suture (Figs. 1, 2, 3 and 4).

The vermilion border of the lip on the outer side of the cleft can usually be traced upward as far as the margin of the nostril. On the surface of the lip, medial to and a little below the ala of the nose, we can distinguish a curved line or groove (Fig. 2, + *C*), with its concavity pointing upward, which reaches from the upper end of the outer margin of the cleft to the ridge on the free margin of the ala which forms the boundary between the nasal mucous membrane and the skin covering the ala. *This groove forms the line of division between the nasal mucous membrane above and the skin of the lip and cheek below.* It is rarely absent. Occasionally its medial end, where it joins the cleft is marked by a slight projection or hump.

If Figs. 1, 2, 3 and 4 be carefully studied the features just described can be verified without difficulty. The attached legends are explanatory: In the photograph (Fig. 1) we can see clearly the semicircular line separating the mucous membrane of the flaring nostril from the skin of the lip, and equally clearly the groove separating columella and philtrum is shown as an oblique white streak. We also see somewhat less distinctly a small triangular area (colored white) on the lower margin of the sound nostril just medial to the ala of the nose, and a similar area just below the ala of the opposite nostril at the lateral end of the semicircular line previously described. In the projection diagrams (Figs. 2, 3 and 4) these features have been accentuated. They are constant enough to justify the following description of the nostrils. "On the affected side the nostril flares widely and the ala of the nose lies at a much lower level than that of the sound nostril. The inner and outer corners of the ala, which for purposes of description I shall call 'horns,' are marked by the letters *A* and *B* (Fig. 2). The rounded end of the ala is shown by a curved line, with its convexity downward, stretching between *A* and *B*. Below the curved line *A B* a triangular area with its apex at *C* can be seen clearly. The line *A C* is prolonged in a

continuous curve until it meets the margin of the cleft at a point marked by a cross, +. The curve is continuous above with the inner margin of the ala. The surface above the curved line belongs anatomically to the mucous membrane of the outer wall and floor of the nose; that below it to the skin of the lip. The triangular area *A B C* is very clearly marked. It is very important anatomically because it represents the triangular prolongation of the lateral nasal process, which in normal development forms the lower margin of the nostril. If the sound nostril be examined carefully a similar triangular area can be seen. It shows white in the photograph. In Fig. 2 this is brought out in relief and shown as the triangle *A′ B′ C′*. The letters on the sound and affected nostrils are placed on similar anatomical points and the triangles are similar in every respect, *i. e.*, *A*, *B* and *C* coincide with *A′*, *B′* and *C′*, and the sides *A B*, *B C* and *C A* coincide with the sides *A′ B′*, *B′ C′* and *C′ A′*. It is quite justifiable to assume that the point *C* represents the apex of the area that was laid down in the embryo to complete the margin of the nostril, and that the curved line *A C* was intended to form the lower margin of the nostril and to lie in the position of *A′ C′* if the nostril had been closed."

In a few cases the features mentioned in the previous description are hard to identify, but as a rule they can be seen without difficulty. Their anatomical arrangement satisfies embryological expectations, and this of itself is a strong argument in favor of the truth of the description. Thus we occasionally see cases of unilateral harelip with the ring of the nostril closed by a narrow tab of integument. If we examine the tab carefully we find that it is triangular in shape, with its base abutting on the ala nasi and its apex attached to the inner margin of the nostril where columella and philtrum meet. It is identical in every respect with the triangular areas previously described and its anatomical features fit in with the embryological requirements.

In Fig. 5, *A*, the normal embryological lines of fusion are shown in the upper lip and cheek. The line (colored red) passes on each side lateral to the philtrum as far as the nostril margin but not into it; thence it courses laterally below the lateral nasal process, around the ala and upward in the groove between the nose and cheek as far as the inner canthus. Between these two lines lie the parts developed from the frontonasal process; namely, the

integument of the nose, including the ring of the nostril, columella and philtrum, and the bony and cartilaginous parts incorporated with them. Lateral to these lines the lips and cheeks are formed by the maxillary process. In Fig. 5, *B*, a condition is shown which is occasionally seen in cases of unilateral cleft of the lip with an intact nostril margin. The rim of the nostril is closed by a triangular tab of tissue derived from the lateral nasal process. The legends are explanatory.

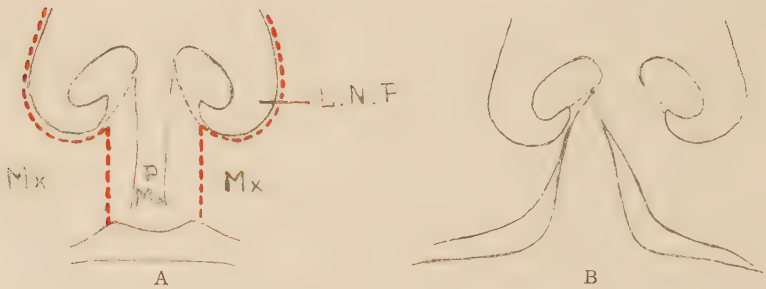


Fig. 5.—The figure *A* shows the lines of fusion between the maxilla and premaxilla by a dashed line, colored red. The triangular areas below each oval nostril, shown by a black dotted line, represent the lateral nasal processes. The ring of the nostril is completed by the union of the lateral and median nasal processes. *L N P* is the lateral nasal process; *M* is the maxilla; *P Mx* is the premaxilla. In the figure *B* an example of incomplete harelip is shown, in which the lower margin of the nostril is bridged over by a triangular tab of tissue derived from the lateral nasal process.

The Alveolar Borders and Hard Palate. The following description is founded on observations made on plaster casts. The figures referred to in the text are either actual photographs of the casts or sketches in which certain important features have been emphasized to make our meaning clearer. We shall study in detail the margins of the cleft, the surfaces of the palatal plates and the alveolar ridges.

The cleft extends from front to back through the whole length of the palate. It divides the roof of the mouth into two areas of unequal size, to the larger of which the septum is attached. The bony framework of the maxilla, which forms the larger area, consists of premaxilla, maxilla, palate bone and septum. We shall speak of it as the *greater process*. That which forms the smaller area, which we shall call the *smaller process*, consists of maxilla and palate bone only (Fig. 6).

The following description will be made clear by reference to Figs. 6 and 7.

THE LARGER PROCESS. On this side the nasal cavity is closed off in normal fashion, as far back as the posterior end of the hard palate, by the fusion of the lower margin of the septum with the edge of the palatal plate and the median cranial ridge of the premaxilla.

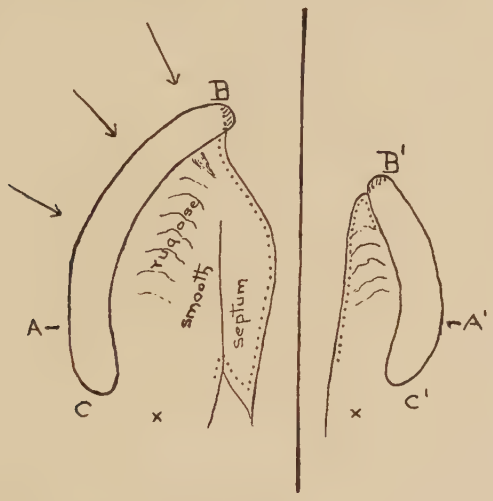


FIG. 6.—Idealized sketch of a plaster mold of a left complete unilateral cleft of the palate. On the left of the figure the palatal surface of the "greater process" is shown. The distorted septum is seen on the border of the cleft. The surface of the true palate shows the smooth and rugose areas. Outside the rugose area lies the alveolar border stretching from *B* to *C*. The three arrows show the direction of the force employed to push the greater process in contact with the lesser during the first stage of the operation. On the right of the figure we can see the palatal surface of the "lesser process," showing the smooth and rugose areas and the alveolar border stretching from *B'* to *C'*. The points *C* and *C'* point to the maxillary tuberosities; *A* and *A'* are on the most prominent parts of the lateral borders of the alveolar processes; *B* and *B'* point to the tips of the alveolar processes. The two crosses × point approximately to the position of the hamular processes.

The Septum. The side of the septum which is exposed to view consists of two separate parts, an upper and lower, which are often separated from one another by a horizontal ridge. The upper hangs vertically in the nasal cavity and is more or less hidden from view in the greater part of its extent by the palatal plate of the "smaller process," the free edge of which almost touches it. In front a large area of this suface can be seen through the flaring nostril. The lower part does not hang ver-

tically, but is bent into a more or less horizontal position and slants gradually toward the edge of the palatal plate of the "greater process," with which it fuses. In some cases the bend is so abrupt that the two parts of the septum lie almost at right angles to each other. The ridge is then very prominent and forms one of the margins of the fissure, to be described later as "the false cleft," which lies between the greater and lesser maxillary processes and which leads from the buccal into the nasal cavity on the side of the lesser process. The horizontal part of the septum in such cases of extreme bending lies almost on the same plane as the surface of the true palate with which it fuses. The line of union of the septum and palate is usually indicated

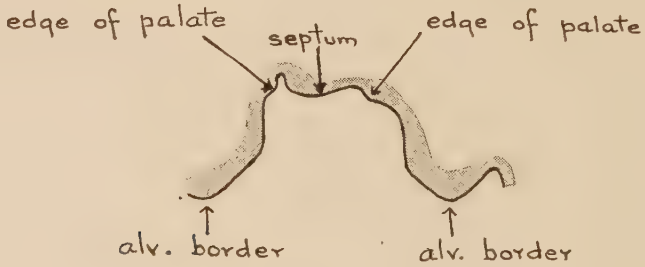


FIG. 7.—Coronal section across a plaster mold of a palate affected with a unilateral cleft. The true cleft is the distance between the edges of the opposed palatal plates, that is, between the points of the lateral arrows. The false cleft is the fissure shown between the bend on the septum and the edge of the palatine plate on the left of the figure. Note the horizontal position of the exposed part of the septum. Note also that the palatine plate of the greater process (right) is more horizontally placed than that of the opposite side (left).

by a slight ridge. It can also be distinguished by a change in the appearance of the mucous membrane, that on the septum being smooth, velvety, elastic and of a deep-red color, while the palatal mucous membrane is dense and tough and somewhat paler in color. The septal mucous membrane is very loosely attached to the underlying perichondrium, from which it can be peeled with great ease in any direction, leaving the perichondrium still attached to the cartilaginous surface. The mucous membrane of the palate, on the contrary, is so closely incorporated with the periosteum covering the palatal plate that flaps lifted up from the hard palate always consist of mucous membrane and periosteum, leaving the underlying bone bare of covering.

The true hard palate lies lateral to the septum. It is bounded in front and laterally by a semicircular mound, called the "alveolar process." Its shape is roughly that of a sector of a circle, the circumference being represented by the alveolar margin. Posteriorly it is continuous anatomically with the soft palate or velum. Its surface is covered with dense mucous membrane closely incorporated with the periosteum covering the underlying bone. The mucous membrane is of two types, one smooth and the other rugose. The smooth area forms a broad strip which occupies the median part of the palate along the margin of the cleft. It reaches from the posterior margin of the premaxilla in front to the velum behind; the rugose forms a narrow strip which lies along the alveolar border outside the former. The rugæ do not usually reach further back than the middle of the alveolar border. The mucous membrane covering the premaxilla is of the rugose type. The posterior portion of the hard palate is covered with smooth mucous membrane which is continuous with that on the buccal surface of the velum. The hard palate ceases at the posterior edge of the palate bone. It is continuous anatomically with the velum, which is a muscular organ covered on both buccal and cranial surfaces with smooth mucous membrane. When cleft, each side is a perfect half of a normal whole, with all its muscles intact and capable of vigorous contractions. The mucous membrane on its buccal surface is continuous around the posterior border with that covering its cranial surface, and this in turn is continuous in front with that lining the floor of the nose. The body of the velum is securely attached to the posterior margin of the palate bone by the palatal aponeurosis, which represents the expanded tendon of insertion of the tensor veli palatini muscle.

The alveolar ridge or border projects from the buccal surface like a mound. It surrounds the front and lateral margin of the hard palate. It consists of bone in which lie developing tooth follicles. It is covered by firmly adherent, dense, elastic, smooth, mucous membrane. The anterior part of the ridge belongs to the premaxilla, the rest to the maxilla. Its anterior extremity projects in front of the anterior end of the cleft.

THE SMALLER PROCESS. This is much smaller than its fellow of the opposite side, because only the maxilla and palate bone enter into its formation. The absence of the premaxilla accounts

for the shortness of its alveolar ridge and the smaller palatal surface. In other respects the general features are identical with those of the larger process. The mucous membrane is of the same character, and the rugose and smooth areas are arranged in the same pattern. The median edge of the palatal process is free, owing to its failure to unite with the septum. It forms one of the margins of the true cleft.

If the two palatal surfaces be compared with one another, it is noticeable that, not only are they different in size, but that they have a different slant, the larger lying more or less horizontal while the smaller is nearly vertical. The smaller palatal plate frequently slants upward so precipitously that its median edge lies opposite the ridge on the side of the septum which marks the boundary between its horizontal and vertical parts. The two structures are, however, always separated by a fissure connecting the mouth and nose. This fissure may be called the "false cleft," to distinguish it from the "true cleft" which is the space between the edges of the opposite palatal plates.

In cases in which the maxillary processes are separated from each other for a considerable distance, and where the septum is greatly distorted, at least two-thirds of the roof of the mouth is formed by the greater process, the buccal surface of which consists of two distinct areas separated by a slight ridge. One lies median to the ridge and belongs to the septum; the other lies lateral and represents the true hard palate. They are easily distinguished from each other by the character of their mucous membrane. Both surfaces lie in the same plane and form a continuous curve which runs across the vault from the alveolar border to the fissure leading into the nasal space which we have previously spoken of as the false cleft. The remaining third of the roof of the mouth is formed by the palatal plate of the lesser process, which often lies in such vertical position that its buccal surface faces the median line (Figs. 7 and 8).

The Cleft. The "true cleft" is the space between the anterior ends of the alveolar borders, in front, and the medial edges of both palatal plates and the vela behind this point. It must be carefully distinguished from the fissure, or "false cleft," which separates the side of the septum from the median edge of the smaller palatine plate. The true cleft varies greatly in width. Between the tips of the alveolar process it is frequently 10 mm.

wide and in extreme cases it may be as much as 15 mm. At the level of the center of the hard palate it is frequently wider by several millimeters than at any other point either in front or behind. The outline of the cleft is consequently somewhat spindle shaped. The false cleft is usually very narrow. It lies to one side of the middle line of the roof of the mouth between the side of the septum and the free edge of the smaller palatal process. In cases where the septum is greatly distorted its lateral position is very much accentuated.

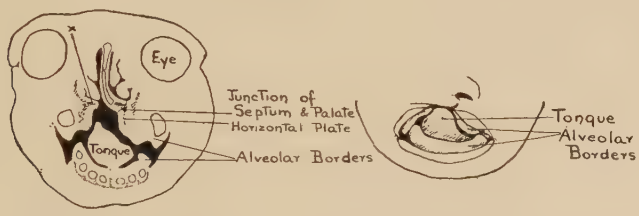


FIG. 8.—Two pictures of the head of a seven months' fetus in which there was a right-sided complete unilateral cleft of the lip, alveolar margin and palate. The figure on the right shows the external appearance of the cleft in the lip. The left hand figure is an accurate drawing of a coronal section of the head through the anterior part of the palate. It shows the relations of the palatal plates to one another and the exact boundaries of the cleft. The site of fusion of left palatal plate and septum is indicated by an acute bend. The lower part of the septum is bent like a hockey stick. Both palatal plates have been pushed into a vertical position. The free edge of the right palatal plate (left in the figure and shown by the letter X) is considerably higher than that of the left. The alveolar border of each maxillary process lies here outside that of the mandible, whereas under normal conditions the alveolar border of the mandible should lie a little outside that of the maxilla. In the actual specimen the tongue occupied the space between the separated maxillæ, and its dorsum (triangular shaped in the section) projected upward between the edge of the left maxillary plate and septum.

The anatomical features just described can be verified by study of the attached figures and photographs. The structure of the roof of the mouth is particularly well illustrated in the coronal section of the head of a fetus (Fig. 8) and that of a plaster cast of an actual patient (Fig. 7).

It must be remembered that the description given refers only to cases of unilateral cleft in which the maxillary processes have been pushed apart for a considerable distance and the septum forms part of the roof of the mouth. In milder degrees of the deformity the roof of the mouth is formed entirely by the palatal plates of the maxillæ and palate bones. The septum lies vertical in the nasal cavity and is barely exposed to view. The

maxillary processes are separated by a narrow fissure, which is the "true cleft," one margin of which is formed by the edge of the lesser palatal process and the other by the line of fusion of the opposite palatal process and the septum. This type of palate is not often met with in cases of complete unilateral cleft of both lip and palate, but it is frequently seen where the fissure is incomplete in the lip or the alveolar border.

In a previous paper I advanced the view that the distortion seen in cleft palate was the result of the pressure exerted by the muscular contraction of the tongue of the fetus. As I have since seen no reason to change my opinion, I submit the original argument for your consideration: "The moment embryonic fusion fails, the great and probably the only agent in preventing subsequent union is the tongue, which exerts constant pressure between the edges of the cleft, until it succeeds, not only in prying the jaws apart, but in pushing the palatal plates upward until they come to occupy a somewhat vertical position. The mechanism of this disrupting force can be clearly traced in every case. Whenever the gap is large enough for the tongue to be inserted, evidences of muscular pressure are always present, and the direction in which the disrupted parts are moved is always that of the tongue thrust, *i. e.*, from behind forward and from below upward. Since the force exerted by the tongue is greatest at the tip and decreases gradually as we pass backward toward the epiglottis, where it reaches its minimum, we should expect to find the deformity greatest where the prying force of the tongue can be exerted to greatest advantage. This expectation is supported by the actual findings. Thus, in clefts confined to the soft palate the muscular action of the posterior part of the tongue is so feeble and ineffective as to be negligible. The slight disrupting force exerted is more than neutralized by the mobility of the muscular halves of the velum. The deformity is consequently very slight. On the other hand, in clefts extending through both soft and hard palates but stopping short at the alveolar margin, the tongue exerts considerable force on the lateral palatal plates of the maxillæ, forcing them upward and outward against the lateral walls of the nose and producing an unusually wide cleft. In addition, the anterior and lateral portions of the alveolar ring are also pushed outward to a considerable extent. But the severest degrees of deformity occur in examples of complete

palatal cleft extending through the alveolar margin into the nostril. Here the tongue has full play and acts like a wedge, driving the maxillæ apart in front so that they swing outward like double doors on hinges which are situated behind near the posterior ends of the alveolar borders" (*C* and *C'* in Figs. 6 and 9).

FIG. 9.—Sketch of a left complete unilateral cleft showing the separation of the maxillary processes. The horseshoe mandibular curve (shaded) has been superimposed on the maxillæ to indicate the degree of the deformity. The figure is described in detail in the text. *C* and *C'* show the maxillary tuberosities. *A* and *A'* represent the most prominent part of the lateral curve. *B* and *B'* are on the anterior ends of the alveolar processes. The crosses behind and medial to the tuberosities show the position of the hamular processes of the pterygoids. The arrow shows the direction of the tongue thrust on the alveolar margin to reduce the deformity.

The degree of displacement of the maxillæ can be measured by comparing the curve of their alveolar margins with that of the mandible, which may be taken as normal. If molds of the deformed palate and mandible are made and placed in a dental articulator they can be studied at leisure. If tracings of the alveolar curves of maxillæ and mandible are superimposed a graphic demonstration of the degree of distortion is obtained. Such a tracing is shown in Fig. 9. The following points can then be verified: The posterior ends of the alveolar processes of the mandible nearly coincide with those of the maxillæ, showing that the latter (maxillary tuberosities) have not been much disturbed by the pressure of the tongue. In front of the tuberosities the alveolar margin of the maxilla lies slightly outside that of the mandible, and the divergence of the curves increases until we reach the tips of the alveolar processes. There is, however, a difference between the two sides. On the side of the greater process the divergence is so great that the tip of the alveolar

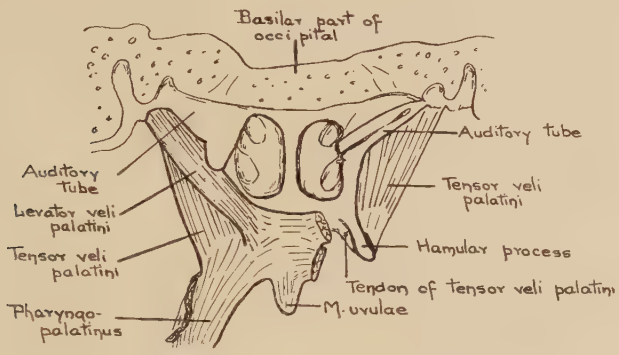
process lies far in front of the mandible, whereas on the side of the lesser process it usually lies along its anterior border. The greater process is always more distorted than the lesser because it stands directly in the way of the tongue as it is protruded from the mouth.

By comparing measurements taken across the palate between corresponding points on the alveolar borders of the maxillæ with similar measurements taken across the curve of the mandible we can measure the extent of the deformity exactly. Three points are taken on each maxilla: at the tuberosities, *C C′*, at the most prominent part of the lateral curve of the alveolar border, *A A′*, and at the tips of the alveolar processes, *B B′*. When they are compared with the mandibular measurements we usually find the following differences: between *C* and *C′* the maxillary measurement is seldom more than 1 mm. greater than the mandibular; between *A* and *A′* the difference may be as much as 2 or 3 mm.; whereas the points *B* and *B′*, which under normal conditions ought to be in contact with one another, may lie as much as 10 to 15 mm. apart.

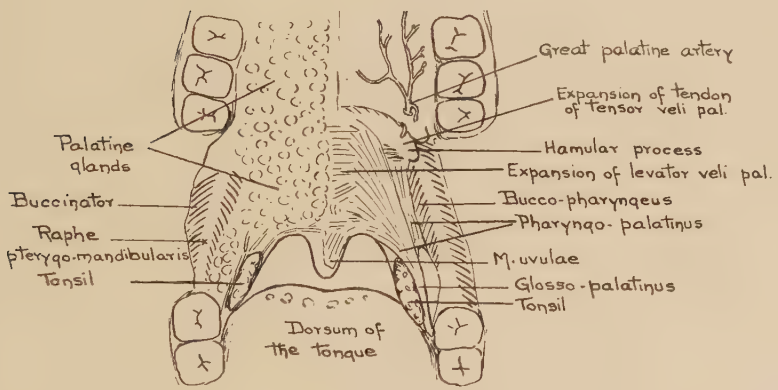
Velum Palati, or Soft Palate. The cleft divides the velum into two perfect halves. The muscles are well developed, and when they contract vigorously, as in retching, the half velum is puckered up into a small mass against the lateral wall of the pharynx. In repose one can see the uvula and the curve of the posterior margin of the velum, which is prolonged into the lateral wall of the pharynx by two prominent folds, called the palatine arches or the pillars of the fauces, between which the tonsil lies. The anterior arch (pillar) covers the glossopalatinus muscle and the posterior the pharyngopalatinus.

It is very important to understand the structure of the velum, and for that purpose the following somewhat detailed description is presented: Its framework is formed by a strong fibrous sheet, called the palatine aponeurosis, which is situated in the anterior part of the velum and whose anterior margin is firmly attached to the posterior edge of the horizontal plate of the palate bone. It forms a common tendon, to which the palatine muscles are attached. The tensor veli palatini is inserted into its lateral part and the rest of the palatine muscles into its posterior margin. The muscles and palatine aponeurosis form the middle layer of the velum. This layer is very thick behind, where it

consists almost entirely of muscles, but thin in front, where muscular fibers are absent. It is covered on both surfaces by mucous membrane, which is stratified on the buccal and ciliated



A



B

FIG. 10.—*A* is a dissection of the palate muscles from the cranial and posterior surface. The origins, insertions and course of the tensor and levator veli palatini are clearly shown. *B* is a dissection of the buccal surface of the palate. On the left the mucous membrane only has been removed. Note the extent and depth of the palatine glands, which form a thick sheet covering the muscles. Also note the pterygomandibular raphé (see text). On the right the palatine glands have been removed and the muscles laid bare. For the description of the muscular layers, see text. Note the relation of the greater palatine foramen to the second permanent molar and wisdom tooth. (Modified from Spalteholz.)

on the nasal side. The buccal mucous membrane is thicker and denser than that on the nasal surface. Mucous glands are very plentiful on the buccal surface over the palatal aponeurosis.

The muscles (Fig. 10) entering into the formation of the velum are five in number, namely, the tensor veli palatini, levator veli palatini, pharyngopalatini, glossopalatini and musculus uvulæ. A short description is necessary: The *tensor veli palatini* arises from the scaphoid fossa and angular spine of the sphenoid and lateral side of the auditory tube. It passes downward between the internal pterygoid muscle and the medial pterygoid plate, and ends in a tendon which hooks around the hamular process and is inserted into the posterior border of the bony hard palate and into the palatal aponeurosis. The palatal aponeurosis is formed mainly by the tendon of insertion of this muscle. It consists of a strong fibrous sheet which is attached in front to the whole width of the posterior margin of the horizontal parts of the palate bones. It serves as a common tendon of insertion for the palatine muscles. Owing to its peculiar mode of insertion the tensor veli palatini has very little action on the velum except to steady the palatine aponeurosis. Therefore, section of this muscle for the purpose of producing relaxation of the soft palate is of very little assistance. The *levator veli palatini* arises from the under surface of the petrous portion of the temporal bone and from the cartilaginous portion of the auditory tube. It passes downward over the upper border of the superior constrictor muscle and enters the side of the soft palate between the two layers of the pharyngopalatinus. Some of its fibers are inserted into the palatine aponeurosis. The rest pass across the middle line and blend with the fibers of the opposite muscle. This is probably the most active of all the palatine muscles. When it contracts vigorously it lifts the velum upward and shuts off the posterior nasal space. Where the velum is cleft it is the strongest of all the muscles in pulling the velum against the pharyngeal wall. The *pharyngopalatinus* lies in the velum and the lateral pharyngeal wall. In the velum it consists of two layers—a superior, which interlaces across the middle line with that of the muscle of the opposite side, and an inferior, which is attached to the posterior border of the hard palate. The levator veli palatini and musculus uvulæ lie between these two layers. The lower pharyngeal fibers lie in the posterior palatine arch (posterior faucial pillar) and blend with the stylopharyngeus muscle anteriorly. They are inserted into the posterior border of the thyroid cartilage and the aponeurosis of the pharynx.

This muscle through its pharyngeal attachment exerts considerable pull on the posterior part of the velum and the side of the uvula. It helps to close the posterior nares by acting in conjunction with the superior constrictor muscle. When it is divided during the course of a palate operation the posterior part of the soft palate and uvula lose their power of contraction. The *musculus uvulæ* consists on each side of a small, narrow bundle of muscle which stretches from the tip of the posterior nasal spine from which it takes its origin to the tip of the uvula where the muscles of each side unite. They are placed side by side and they lie along the levator palati between the two layers of the pharyngopalatini muscles. The *glossopalatinus* is a thin muscle which lies in the anterior palatine arch (anterior faucial pillar). It is attached in the velum to the palatine aponeurosis; in the tongue it blends with the styloglossus and the intrinsic muscles of that organ. In the velum it lies just under the mucous membrane and covers the anterior layer of the pharyngopalatinus.

It will be seen from the description that the muscles mainly responsible for lateral traction on the velum are the levator veli palatini and the pharyngopalatini. The tensor probably does nothing more than hold the palatal aponeurosis tight, in order that it may serve as a rigid support to the other muscles.

Nerve Supply to the Palate Muscles. The tensor veli palatini is supplied by a branch from the fifth cranial nerve through the otic ganglion. It enters the muscular belly of the muscle as it lies between the two pterygoid plates. It is never in danger of division in palate operations. The levator veli palatini is supplied by twigs derived from the pharyngeal plexus. It seems probable that these fibers are primarily derived from the accessorius and that they reach the plexus through the pharyngeal branches of the vagus. They are very hard to dissect. They appear to enter the belly of the muscle at intervals at several points between its origin and where it enters the substance of the velum. It seems probable that if the muscle were divided near its origin, just anterior to the auditory tube, it would receive very slight permanent damage because the nerve supply to its distal part is not divided. We have verified this assumption in our operated cases. We have found that the divided ends of the muscle unite by scar tissue, which resembles a tendinous intersection of the rectus abdominus; and that afterward, when heal-

ing is complete, the function of the muscle is restored. All the rest of the muscles of the soft palate are supplied by twigs from the pharyngeal plexus which are very difficult to trace. The twigs to the palatoglossus and musculus uvulæ reach their destination by coursing along the pharyngopalatinus and levator palati.

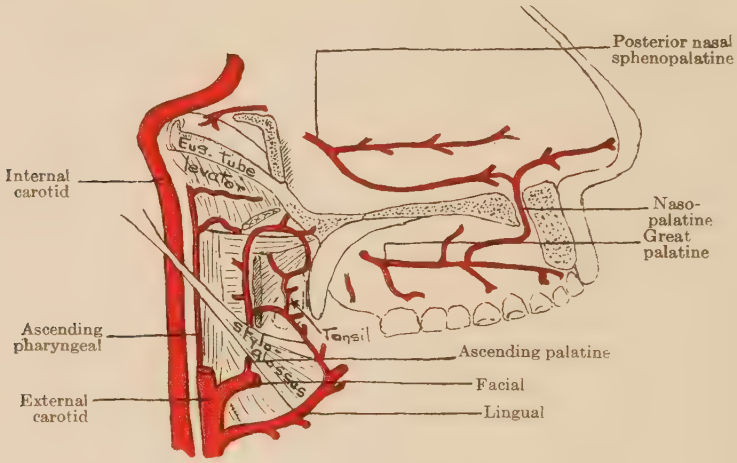


FIG. 11.—Diagram of the arteries supplying the palate. For description, see text (Modified from Spalteholz.)

Blood Supply to the Palate and Velum (Fig. 11). It is well here to consider the blood supply of the whole palate:

1. From the descending palatine branch of the internal maxillary artery we have the following branches: (*a*) The greater palatine artery which emerges from the greater (posterior) palatine foramen, which lies just in front of the hamular process. This branch runs forward close to the alveolar margin and anastomoses in front with the nasopalatine, which reaches the palate through the foramen incisivum. It supplies the whole length of hard palate. (*b*) Several lesser palatine arteries, which emerge from the "foramina palatina minora" and are distributed to the velum.

2. From the posterior nasal (septal artery) we have a small branch which reaches the front of the palate by passing through the foramen incisivum.

3. The ascending palatine artery is derived from the external maxillary (facial). This passes into the substance of the velum

along the levator veli palatini muscle and anastomoses in front with branches of the lesser and greater palatine arteries, and behind with the tonsillar branch of the external maxillary and with tonsillar branches of the dorsalis linguæ.

4. The ascending pharyngeal artery gives off pharyngeal branches which reach the auditory tube and tonsil and ramify with the branches just described in 3. Sometimes it gives off a special branch (palatine) which replaces the ascending palatine and passes into the velum along with the levator palati muscle.

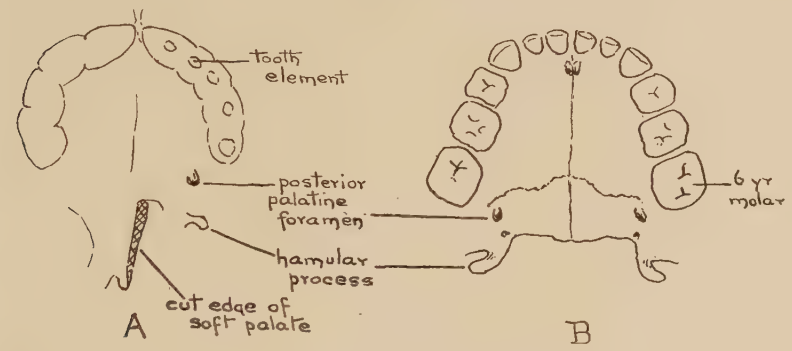


FIG. 12.—*A* is a sketch of the palate of a fetus at term showing the alveolar processes (mounds) containing the tooth elements of the temporary teeth. Note the relationship of the posterior palatine foramen to the second premolar tooth and to the maxillary tuberosity; also note the constant relation of the hamular process to the foramen, about 6 mm. behind it. *B* is a sketch of the palate of a child of about seven years of age, with an erupted six-year permanent molar tooth. Note the relationship of the posterior palatine foramen to the tooth. Note also that the distance between the palatine foramen and the hamular process has increased slightly (about 8 mm.). Compare this with Fig. 10 *B*.

The relations of the greater palatine foramen, hamular process and tuberosity of maxilla to one another are of great importance (Fig. 12). In babies the alveolar border of the maxilla is not developed further backward than the level of the second premolar tooth. It terminates in the maxillary tuberosity, behind which the under surface of the maxilla can be palpated as far back as the hamular process. There is quite a considerable space between the tuberosity and the hamular process, which is covered by mucous membrane which in reality belongs to the hard palate. The greater palatine artery enters the palate just in front of the hamular process, and the relation of the tip of this process to the greater palatine foramen, from which the artery

issues, remains unchanged during the life of the individual, the increase in distance being just what one would expect from the increase in the size of the bones forming the palate. The arch of the palate in babies is usually shallow and, consequently, the line of the greater palatine artery, which courses directly forward in the direction of the foramen incisivum, seems to lie further

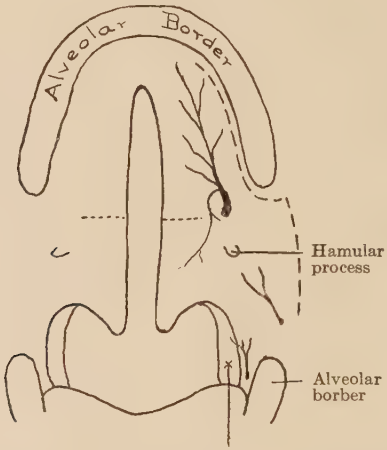


FIG. 13.—Diagrammatic sketch of a palate, after the repair of the alveolar border and the anterior part of the palate, as it appears just before the final operation for closure of the posterior part of the cleft. The mouth has been opened so wide that the tuberosities of both maxillæ and mandible can be seen. The velum has been purposely represented longer than normal in order to emphasize its anatomical structure. The greater palatine artery is seen in front, passing forward in the substance of the hard palate. Note its recurrent branch passing backward into the fleshy velum. Arborizing twigs are seen behind the hamular process, derived from the ascending palatine and tonsillar branches of the external maxillary artery. The dashed line coursing backward along the medial border of the alveolar process and passing around the tuberosity of the maxillary and thence toward the mandibular tuberosity is the lateral incision frequently used to liberate the flap from its lateral connections (see text). The position of the hamular process is approximately correct. The posterior border of the palate bone is represented by two dotted transverse lines on each side of the cleft.

away from the alveolar border than it does in the palate of older people, in whom the palatal plates frequently lie in an almost vertical position. As age advances the tooth germs of the permanent molars push toward the surface and the alveolar margin develops backward, until, when dentition is completed, the tuberosity of the maxilla projects backward beyond the greater palatine foramen. In adult skulls with complete eruption of all

the molar teeth the foramen may lie opposite the wisdom tooth or even further forward (Fig. 10). The distance between the hamular process and the palatine foramen varies from 6 mm. in the skulls of children to 12 mm. in the skulls of adults.

It is evident from this description that in children lateral palatal incisions can be carried along the whole length of the alveolar border, from just outside the os incisivum in front to the tuberosity of the maxilla behind, without endangering the arterial supply of the hard palate (Fig. 13) and, further, that the incision can be prolonged considerably in a backward direction if its posterior end is curved around the tuberosity and the line of the cut is kept well outside the tip of the hamular process. If the mouth is opened widely with a gag, a sharp ridge can be seen under the mucous membrane which stretches from the tuberosity of the maxilla to that of the mandible. This indicates the direction of the line of incision. If the mucous membrane over this ridge is divided a fibrous band is exposed. This is the pterygomandibular raphe, which is an aponeurotic intersection between the buccinator muscle in front and the superior constrictor of the pharynx behind. If an incision is made in the raphe from maxilla to mandible the lateral supports of the velum are severed, and it is an easy matter to push the velum, faucial pillars and tonsil of this side bodily toward the middle line without hurting the arteries which reach the palate from below.

Repair of the Deformity

In plastic work on the lip and palate one cannot emphasize too strongly the wisdom of operating in stages. We invite failure by attempting too much at one sitting. Failure of union may make a subsequent operation difficult and hazardous, while sloughing is so disastrous that the case may be irretrievably ruined. It may be laid down as an axiom that the final result depends more on the proper sequence of operations than on any other factor except infection. In a former paper I presented a table in which an attempt was made to standardize the sequence and character of the successive operations required to close lip and palatal defects. I have found it so valuable that I venture to present it again for your consideration.

TABLE OF ALTERNATIVES

Operation.	First alternative.	Second alternative.	Third alternative.	Fourth alternative.
First . .	Lip and alveolar border	Lip, alveolar border and anterior part of palate	Alveolar border and anterior part of palate	Alveolar border.
Second . .	Palate, complete at one sitting (rare) or anterior part only	Palate, posterior part	Lip	Lip.
Third . .	Palate, posterior part	....	Palate, posterior part	Palate, complete at one sitting (rare) or anterior part only.
Fourth . .	.....	....	.....	Palate, posterior part.

This table was drawn up as the result of my experience during many years of activity, and it is a mirror of the evolution of my lip and palate operations during this period. The table is easy to read, and if one believes, as I do, that the deformity of the alveolar border should be repaired early in life, it can be applied to almost all of the typical examples of complete unilateral and bilateral clefts of the lip and palate. For atypical cases, which are usually a law unto themselves, a special table of sequences must be drawn up to meet the individual peculiarities of each case. To understand the method of applying the table, let us take as an example a typical case of complete unilateral cleft of the lip and palate with a reasonably wide cleft. If we choose the sequence of operations outlined under the first alternative three operations will probably be needed to close the defect. At the first operation the alveolar border is restored and the lip repaired; at the second operation the anterior part of the palate and at the third the posterior part of the palate. The other alternatives are read in the same manner, to wit: In the second, two operations; in the third, three; in the fourth, four. After a little experience the proper sequence is chosen with the least margin of error and the final results improve correspondingly. The first three alternatives are particularly applicable to complete unilateral clefts, whereas, complete bilateral clefts usually fall into the fourth. Considerable latitude can be taken with the first alternative. Thus, at the first operation the lip

alone can be repaired, and if sufficient time is allowed to elapse between the first and second operations, the anterior ends of the alveolar ridges will come together spontaneously and the cleft will be narrowed considerably. By this means we avoid molding and penetration of the jaw with the silver-wire suture, and so avoid the risks of injuring the tooth follicles. The subsequent operations follow according to the table. The second alternative is somewhat risky. It should never be attempted unless the child is in splendid physical condition. The first operation of the sequence is very extensive and is usually followed by severe shock. The third alternative is, in my opinion, the operation of choice in all of the cases of complete unilateral cleft in which the septal flap is used.

The following description is a faithful account of the operative steps required to close a typical example of this deformity.

First Stage: Closure of the Alveolar Cleft and the Anterior Part of the Palate

Steps and Technic of the Operation. The object of the operation is to restore the curve of the alveolar border and to close the anterior part of the palate by means of a flap taken from the side of the septum. This constitutes the first stage in the sequence of operations comprised in the third alternative. The lip and posterior part of the palate are not touched at the first sitting. If the operation is successful we secure union of the anterior ends of the alveolar process and a third or a half of the front of the palate. Furthermore, we seldom fail to secure continuous union from front to back and consequently avoid the troublesome holes and fissures which so frequently follow operations that pay no attention to the coaptation of the alveolar processes.

An incision, the outline of which is shown in Fig. 14, is made on the side of the septum. It courses horizontally from front to back along the ridge which separates the vertical and horizontal parts of the septum from each other. The anterior end of the incision is carried forward into the nasal hollow which overhangs the premaxilla, and then curves sharply around toward the anterior end of the groove which separates the alveolar ridge of the premaxilla from its palatal surface. It stops at

this point. The posterior end of the incision is carred backward until it reaches the posterior border of the septum, which it follows medially to its junction with the hard palate. The incision outlines a flap on the septum whose free edge is considerably larger than its base. The base is formed by the junction of septum and mucoperiosteal layer of hard palate, where the two structures are anatomically continuous. Its shape is roughly that of a mushroom (button), with sharply cut corners. The flap, which consists of mucous membrane only, is now peeled

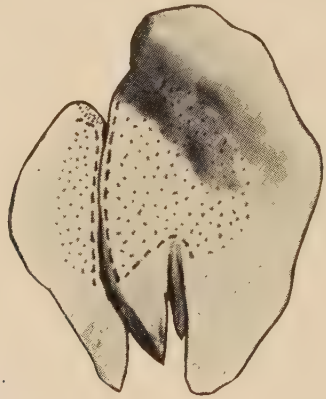


FIG. 14.—Photographs of a plaster cast of a palate, showing the incision employed in raising the septal flap and the area of mucoperiosteum lifted up from the palatal plates. The dashed lines show the incisions and the dotted areas the extent of the undermining of the palate. On the right of the figure the extent of the flap lifted up from the septum and palate is shown. On the left, over the "lesser process," a much smaller area is lifted up. The whole palatal plate on each side is denuded of its mucoperiosteal covering. An oval-shaded area is seen on the tip of each alveolar process. These areas are denuded of their mucous membrane and brought in contact with each other when the maxillæ are approximated.

from the septum with a periosteal elevator and reflected toward its base. It is handled very carefully and retracted by means of two fine silk threads passed through its corners. It peels up with great ease as far as its base, where it blends with the hard palate. Along this line a curved elevator is passed under the mucoperiosteal layer of the hard palate. It is then quite easy to peel the mucous membrane of the hard palate from front to back and outward as far as the alveolar border. Behind, where the velum and hard palate blend, it is very difficult to raise the flap from the underlying bone, because the palatal aponeurosis

(the tendon of insertion of the tensor veli palatini) is firmly attached to the posterior edge of the palate bone. If a periosteal elevator is used in this situation there is great danger of tearing the flap. It is better to sever the attachments in the following manner: Pass a traction suture of fine silk through the velum, pull on the traction stitch and expose a slight area of the cranial surface of the velum and sever the attachments of the velum to the posterior edge of the palate bone by successive snips of curved scissors or by short cuts made with the edge of a curved Lane knife. The structures divided are nasal mucous membrane and palatal aponeurosis. This part of the operation must be done with extreme care and precision, for it is very easy to puncture or tear the thin flap just in front of the fleshy velum unless the cutting instruments are kept continuously in contact with bone. There is practically no danger of wounding the posterior palatine arteries unless the dissection is carried too far backward and laterally. As soon as this stage is reached the whole palate, with the septal flap attached to it, hangs free on its lateral and posterior attachments. The dotted area on the right side of Fig. 14 shows the outlines of the flap.

We now turn our attention to the palatal surface of the smaller maxillary process on the other side of the cleft (Fig. 14, left side). The periosteal elevator is inserted at the free edge of the cleft between the bony palatal plate and its mucoperiosteal covering, and a flap of mucoperiosteum is elevated from the underlying bone over its whole area. The extreme anterior end of the flap is thrown up as a small tongue by carrying an incision backward for a short distance along the groove separating it from the alveolar border. It is also handled by traction sutures passed through its free edges. Care must be taken to liberate it freely, in order that its raw surface may be brought in contact with that of the septal flap without distortion or tension. To this end it may be necessary to divide on this side also the palatal aponeurosis and nasal mucous membrane from the posterior edge of the palate bone. When this is done the whole palate will hang freely on its lateral and posterior attachments. This completes the first stage.

The technic of the operation just described differs materially from that used in our early work on the septal flap because at first we were afraid to lift the flaps up widely from the underly-

ing bone. We soon found, however, that unless the flaps were freely movable it was embarrassing to pass the sutures and difficult to get approximation without tension and puckering. Therefore, we started to lift up the mucoperiosteal flaps over a larger and larger area until we have now arrived at a stage where we separate the entire hard palate from the underlying bone as a routine practice. We also found that it was very difficult to bring the anterior ends of the flaps into good apposition unless the front of the hard palate was well mobilized. This was accomplished by carrying an incision about 1 cm. long backward from the margin of the cleft along the groove separating the alveolar process from the hard palate. This enabled us to lift up the front of the hard palate as a tongue-shaped flap and to bring its raw surface well into view. This maneuver may not be needed on the side to which the septal flap is attached, if the flap is long and mobile, but it is always necessary on the opposite side of the palate.

In our early operations we frequently made use of a side incision (Langenbeck) along the alveolar border of the hard palate, to which the septal flap was attached to enable us to lift it up more easily. We found that it was harmful, inasmuch as it allowed the hard palate medial to it to become puckered and narrowed during the healing processes and was often followed by an excessive formation of scar tissue that made the subsequent operation on the posterior part of the palate more difficult. Therefore, we seldom use it now, except in cases where denudation from the side of the cleft is impossible. Deliberate division of the palatal aponeurosis was first done in several cases where tension on the flaps would otherwise have been unavoidable. The results were so good that we have since employed it without hesitation whereever we have found it necessary. It does not appear to injure the velum or to distort it in any way. In a short time the mucoperiosteal layer of the hard palate settles down on its bony bed and grows firmly to it. At a later date, while closing the posterior part of the palate, we find the posterior part of the hard palate and velum very little changed for the worse, either in structure or position.

The flaps are now ready for approximation. Certain preliminary steps are necessary, however, before the sutures are inserted.

Molding the Maxillæ. The maxillæ are usually so far apart that it is seldom possible to bring the flaps together without tension; therefore, it is necessary to push the bones together and mold them into shape. In young babies, up to the age of three or four months, the molding can be done by thumb pressure, but in older children the bones may have to be divided with a chisel. If thumb pressure alone is used the force is directed at first against the front of the "larger process" at a point lateral to the premaxilla, in order to drive the maxilla bodily toward the middle line and narrow the cleft (Fig. 6). Pressure on the premaxilla must be carefully avoided until the main part of the greater process yields and slides toward the opposite side. Pressure is exerted on the outside of the cheek above the line of the alveolar border, in order to avoid bruising or breaking this delicate process in which the tooth elements are embedded. With perseverance and patience the bone moves over gradually into the desired position. No attempt is made to disturb the posterior connections of the bone, because, as we have previously pointed out, the distance between the tuberosities of the maxillæ is seldom increased more than 1 mm. Pressure is kept up until almost all the natural resilience of the bone is destroyed and there is no longer a tendency for it to spring back into its former position. During this manipulation no attempt is made to alter the position of the "lesser process." It is merely steadied by the thumb of the opposite hand. If the molding is successful the premaxilla will occupy the front of the curve of the restored alveolar border and the tips of the alveolar processes will touch one another. The natural forward projection of the premaxilla must be preserved. If it is pushed too far backward a flat upper lip will result (Fig. 15). The areas of contact on the tips of the alveolar processes are now denuded of mucous membrane in order that they may unite when they are brought together.

Suturing the Maxillæ. A suture is now passed through the maxillary processes in order to hold them together. The bones are penetrated at a considerable distance above the alveolar margins in order to avoid injury to the tooth sacs. At the level of the tooth sac of the second premolar, or thereabouts, a small incision is made through the mucous membrane at the junction of lip and gum, and the cheek is lifted up from the outer surface of the maxilla over a small area. A curved brad-awl is then

driven through the bone toward the middle line in such a direction that its point keeps above the level of the bony palatal plate. The palatal flap is carefully lifted out of harm's way. As soon as the point of the instrument shows in the cleft it is withdrawn. A curved Emmet needle threaded with a loop of strong silk is now passed along the track of the brad-awl and carried through into the cleft and withdrawn by way of the mouth. The loop of silk is left in place. A piece of strong silver wire is now bent into the silk loop, which is withdrawn, carrying the wire with it backward along the bony channel made by the brad-awl. The wire is left in place, one end sticking out from under the lip and the other from the cleft. The same

FIG. 15.—Photographs of casts of a case of unilateral cleft of the palate before and after closure by the septal flap. On the left the picture shows the palate before operation. Note the almost horizontal position of the lower part of the septum. On the right we see the palate at the age of fourteen months, one year after the first operation, just before the operation on the posterior part of the palate. Note that almost the whole length of the hard palate is closed and that the premaxilla lies in good position.

maneuver is repeated on the other side, and the end of the wire projecting from the cleft is drawn through the bone. The two ends of the wire now lie in front of the corresponding maxillary processes, while the loop lies in the nasal cavity above the palatal processes. Each end of the wire is now carried forward on its own side between the cheek and the bone toward the cleft in the lip, which it enters at the level of the flaring nostril. The wire thus passes in front of the premaxilla without penetrating it, and traverses each maxilla at the same point—that is, about the level of the second premolar. The front part of the wire is completely buried under in the soft tissues of the lip and cheek, except where it lies in the cleft below the nostril. The

loop (bight) lies in the nasal space (Fig. 16, *A* and *B*). The slack of the wire is taken up, but no attempt is made to tighten it at this stage. The ends are left long and anchored.

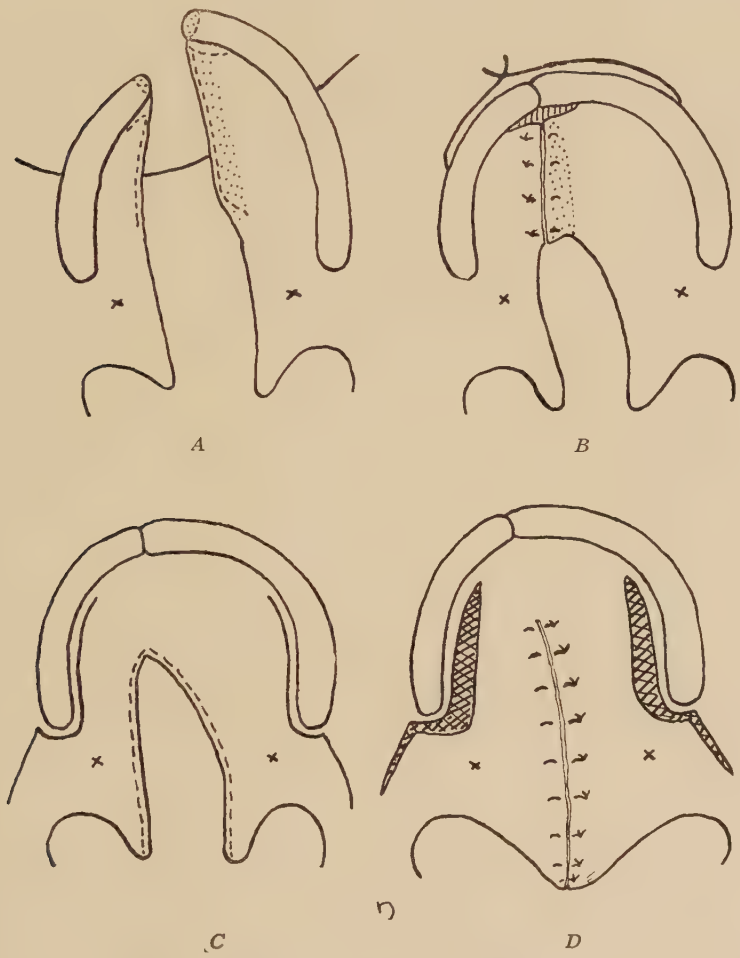


FIG. 16.—Shows the various stages of the repair of the palate. *A* and *B* the method of closing the front of the palate by means of a septal flap. In *A* the outline of the flap is shown and the silver wire transfixing the maxillary processes. In *B* the maxillary processes are approximated, the silver wire is twisted in front, and the palatal flaps are fastened together with four mattress sutures.

C and *D* show the stages of the second palatal operation. In *C* the side incisions and extent of denudation of the fissure are shown. In *D* the edges of the cleft are fastened together with a row of mattress sutures. The side incisions are showed as gaping slits. The crosses are over the hamular processes.

Approximation and Suture of the Palatal Flaps. At this stage the sutures should be passed through the palatal flaps. The anterior end of the septal flap is approximated to the anterior end of the palatal flap on the opposite side of the cleft and the edges of the two flaps are applied to each other. One can then see where the stitches should be placed. The posterior stitch is passed first and the rest *seriatim* from behind forward. It is seldom that more than four stitches are needed; as a rule, we use three only. All are of the mattress type and they are inserted in such a manner that broad strips of the raw faces of each flap

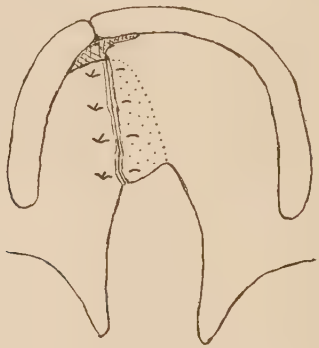


FIG. 17.—Sketch of the appearance of the palate when the maxillæ are approximated and the flaps fastened together. The tips of the alveolar processes are in contact. Behind them a shaded area is shown which designates the denuded bony surface of the premaxilla and of the lesser palatal process. The line of fusion of the septal flap with the mucoperiosteal flap of the greater process (right) is shown by a dotted line. The vertical mattress stitches are inserted and knotted but not tied tightly, consequently the flanging of the area of apposition of the flap is not shown. No attempt has been made to obtain perspective and therefore the septal flap appears much narrower in front than it is in reality.

will lie in contact when the stiches are tightened. The sutures are shown in Fig. 16, *B*, and Fig. 17. We usually speak of this form of suture as the vertical mattress stitch. We have found that it does not cut off the blood supply to the edges of the flap and, as a result, that flaps fastened together in this way seldom fail to unite. Fine silkworm gut is used as suture material. The sutures are passed and anchored in position, but not tied at this stage.

Approximation and Fixation of the Maxillæ. The maxillæ are now pushed firmly in contact with one another until the raw areas

on the tips of the alveolar margins are in contact. The slack of the silver-wire suture is then taken up and the free ends are twisted firmly together. During this maneuver the palatal flaps are held carefully out of the way by the sutures.

Tying of the Sutures in the Palatal Flaps. The palatal sutures are now tied, beginning behind and finishing in front. If necessary a few supplementary stitches of fine silk are placed between the mattress stitches. The mattress stitches must not be fastened too tight. We need firm approximation of the sides only. When all the stitches are tied the line of suture resembles that of the flanges of a steam pipe (Fig. 16, *B*, and Fig. 20). Finally, the sutures are cut short and all the traction stitches which hitherto have been used to hold the flaps in position are removed. The silver-wire suture projecting from the nostril is now cut distal to the twisted knot, leaving the ends about $\frac{1}{4}$ inch long. This completes the operation.

After-treatment. The after-treatment is very simple. The flaps are never touched or interfered with in any fashion. Water is given immediately and milk feeding allowed the same day. The silver-wire suture through the maxillæ is removed in two or three weeks. The sutures in the palate usually come away spontaneously. If necessary, we remove them in four or five weeks. The subsequent operations depend on the growth and health of the child.

The lip is repaired as soon as the mouth is clean. This may take six weeks or two months. An interval of about six months is allowed to pass before the posterior part of the palate is repaired. We prefer the child to be twelve months old, although occasionally we operate in robust babies when they reach nine months. The best guides are the weight and the general health. A baby of twelve months who has good digestion and weighs 20 pounds is a safe risk.

At this age inspection of the palate will show that great changes have occurred since the first palate operation (Figs. 15 and 16, *C*). The ends of the alveolar processes are either in close contact with each other or are united by firm scar-tissue. As a rule, the anterior half of the palate is completely closed. The septal flap has accommodated itself to its new position and the mucous membrane has lost its fine velvety texture. The line of union between the edges of the septal and palatal flaps is shown by

a prominent ridge. Behind the restored palate the cleft is invariably much narrower than it was at first, not relatively merely, but actually. Sometimes it is so narrowed that the edges of the vela lie almost in contact with one another when the muscles are at rest. The consistence and suppleness of the vela are very slightly changed from that of an untouched palate. Occasionally they are a little thicker and more edematous, but real scar-tissue is seldom present. The posterior part of the mucoperiosteum of the hard palate always reattaches itself to the underlying bony plate after the first operation, and, as far as we have been able to make out, the palatal aponeurosis is as firmly attached at the second operation as it was at the first.

The immediate results obtained by the operation have been very satisfactory. If the babies are well nourished shock is seldom seen. In fact, the operation does not seem to be more severe than repair of the lip and nostril alone. We are very careful, however, to choose fit cases and, if necessary, to postpone operation until the baby is in good condition. The best time for operation is somewhere about the second or third month. The bones are still soft and can be molded fairly easily. At the age of six months the bones are frequently so hard and unyielding that they have to be divided with a chisel before they can be brought together. I have occasionally operated successfully on babies less than a week old. On calm reflection, however, I think that such early operations should not be advised, because they serve no particular purpose. They are more difficult than later operations and they do not enable the baby to suckle from the mother's breast. In no respect can we regard the operation as life-saving. It is merely one of convenience and, therefore, can be postponed until the baby is healthy enough to justify it.

Our experience in the use of the septal flap deliberately, as an integral step of the operation, does not date further back than six years at the utmost. Previous to this time we had performed the operation occasionally, in a somewhat timid fashion, as a help when closing the front of the cleft in cases where the lip alone had been repaired at a former operation, but where no attempt had been made to bring the maxillæ together. In such cases the cleft was so wide in front that closure by a true palatal flap

was invariably followed by the persistence of a hole just behind the alveolar border. Although we could confidently expect the ends of the alveolar border to come closer together as the years passed, this hole still persisted and was very troublesome to close. At this time we were operating on these cases almost always by the sequence of operations shown in the first alternative (see table), in which at the first operation the maxillæ were approximated and fastened together with a strong kangaroo-tendon suture and the lip was repaired at the same time. The suture was knotted in front and covered by the repaired lip. It was allowed to remain in place until it was absorbed.

By degrees we began to pay more attention to the approximation of the tips of the alveolar processes and then to the front part of the palate, which we began to close by a septal flap, taken somewhat timidly at first, but more boldly later on. Finally the operation assumed the magnitude of a complete repair of the lip, alveolar border and front of the palate in one sitting, as shown in the first operation of the second alternative. We found, however, that this was too severe for the average baby, and consequently we stopped short after the repair of the palate and left the repair of the lip to a subsequent operation. For a time we still used the kangaroo tendon to hold the jaws together. In several cases, unfortunately, the tendon was absorbed too quickly. It broke away in about a week's time and allowed the maxillæ to separate. In one instance the palatal flaps were torn apart. Since that time we have used silver wire as a routine practice. It is left in place for a month or more, after which period there is no tendency for the bones to move apart.

The great objection to our method of operating lies in the risk of injury to bone and especially to the tooth follicles during the passage of the silver-wire suture. We try to avoid the tooth follicles by drilling the maxillæ far away from the alveolar margin, but they lie so high in the alveolar processes under normal conditions that they are injured occasionally in spite of all our care.

It is too early yet for me to speak authoritatively about the condition of the permanent teeth in the patients that I have operated upon; but we may safely infer, from the available observations on dentition following operations in which the jaws have been penetrated by silver wire, that eruption will be abnor-

mal in some of our cases. There is no doubt that dentition is far more regular in maxillæ that have not been disturbed. It is a matter of common observation that cases of cleft palate which have not been operated upon in infancy usually show remarkable regularity of the teeth except in the immediate neighborhood of the fissure. And, furthermore, careful observations have shown that mere closure of the palate by the Langenbeck method, without injury to or disturbance of the bone, if performed in the early years of life is frequently followed by narrow palates, distorted alveolar borders and irregular dentition. On these findings some surgeons, logically enough, base their argument that late operations on the palate are invariably followed by better anatomical results than early ones. They advise repair of the lip early in life, but do not touch the palate until the children are five or six years of age. During this time the maxillæ undergo spontaneous molding by the pull of the lip and the contraction of the facial muscles, and the cleft becomes so much narrower that in many cases the tips of the alveolar processes lie in contact. Repair is then easily accomplished without danger of injury to bone or tooth follicles.

We have always believed that patients whose palates have been repaired before they learn to talk, speak better than those who have learned to talk before the repair. We feel certain that patients whose palates have been repaired at the age of a year, or even as late as eighteen months, speak much better than those in whom we have repaired the palate at the age of five years or later, and consequently we have been encouraged to persist in our methods. We feel that the advantages of the early operation outweigh its disadvantages, and that our results so far are much superior to those we have obtained hitherto. If we felt otherwise there would be no justification for our teaching except the sentimental desire to efface the repulsive deformity and make the unfortunate children presentable as early as possible.

Second Stage: Repair of the Lip and Nostril

This, the second operation of the series, is undertaken about two months after the operation on the alveolar border, or at the discretion of the surgeon as soon after the first operation as the mouth and nose are clean and dry. The operation we prefer

will be easily understood by referring to Figs. 2, 3 and 4, used as the basis of the anatomical description which has gone before. We always use screw compasses to make the necessary measurements. Punctures are made in the skin with the sharp points to indicate the beginning and end of each incision. A hypodermic needle dipped in methylene blue or other coloring matter is a surer and more permanent way of bringing the punctures into relief. First, we determine the depth of the future lip by measuring from the lower margin of the intact nostril to a point on the vermilion border just lateral to the philtrum (Fig. 2, red line). While making this measurement the edges of the cleft are held together and the lip stretched so as to make temporarily a lip of proper depth. The compasses are then fixed by the screw to prevent movement of the points. Then the following landmarks are pricked out: On the medial side of the cleft the point, C', is taken (Fig. 3) as described previously in the text. It lies on the edge of the cleft, where columella and philtrum meet. Sometimes it is indicated by a slight projection. A deep prick is made here with a sharp point of the compasses. One end of the compasses (fixed previously) is placed on C' and the other end on the vermilion border of the lip. Another prick is made here, which is shown in the diagram at D'. On the other side of the lip the same maneuver is carried out. A prick is made at C (*vide* anatomical description) on the margin of the nostril and another at D on the vermilion border. By this means we have two distances on each side of the cleft of equal length, each one measured from a point (C or C') which will eventually lie on the margin of the reconstructed nostril to another point (D or D') which will eventually lie on the line of the vermilion border of the lip. Taking another pair of compasses, a measurement is taken from the point D' on the vermilion border to a point on the free margin of the lip represented by E'. A deep prick is made at this point. This measurement must always be a little longer than the shortest distance across the mucous border at this level, and consequently the line $D'E'$ usually slants upward toward the nostril, forming an angle of about 60 degrees with the line $D'C'$. A similar point, E, is pricked on the opposite mucous margin. If the points C, D and E and C', D' and E' are joined by lines with the same curve the line CD will be equal to $C'D'$ and DE equal to $D'E'$. If the

area of lip between the margin of the cleft and these lines is removed on each side, and the point C brought in contact with C', D with D' and E with E', the lines $C\,D$ and $D\,E$ will lie in contact with $C'\,D'$ and $D'\,E'$. In addition, the ring of the nostril from C (through A) around to C' will be restored, the lip will be of the proper depth as far as the vermilion border, and the depth of the mucous border will be a little deeper than normal.

By this method we can make a satisfactory lip, but it will fail completely to restore the nostril and to bring the ala nasi into its proper place. The inner horn of the ala (marked A) is brought into its proper position, but the outer horn (marked B) is not brought far enough upward and inward. The line $A\,B$, instead of facing inward in the direction of the arrow shown at $A'\,B'$ (Fig. 2), faces downward and very slightly outward. The flare of the nostril is not fully corrected and the curve of the ala is quite dissimilar to that of the sound side. How can this be remedied? The problem to be solved is to bring the outer horn of the ala, B, inward, and to curl the nostril so that the line of the ala nasi, $A\,B$, will face the middle line of the restored upper lip at the same angle as $A'\,B'$. Turning our attention to Fig. 4, let us assume that the measurements $C'\,D'$ and $D'\,E'$ are satisfactory. The lines $C\,D$ and $D\,E$ are identical with those in Fig. 3. Approximation of the side $C'\,D'$ fails to bring the point B far enough inward and upward. Therefore, the curve of $C\,D$ is unsatisfactory. In the diagram (Fig. 4) two other curved lines have been drawn (on the left), each beginning at C and ending on the vermilion border. Each line is equal in length to $C\,D$. The curves of each of these lines approaches a little nearer to the point B. If it is possible to regulate the curve so that we can fix a point on its convexity which will lie at the proper distance from B, and also at the proper distance from C, then approximation of this curved line with the line $C'\,D'$ will bring this point over to the sound side and will carry the point B inward and upward, so that the line $A\,B$ will correspond in position to the line $A'\,B'$. In plain language, we must undercut the ala nasi in order to restore its curve and bring it into its proper position.

In a previous paper I have described a method of measuring the degree of undercutting, so that the ala would be carried into

the desired position without mistake. I found this to be too complicated for practical purposes. Therefore, in actual practice we guess at the degree of undercutting, but regulate the length of the curved incision by using a piece of lead wire cut to the exact length and curved to our will.

While the measurements are being made the lip on each side of the cleft is steadied by traction sutures which are placed on each margin of the cleft inside the nostril just above C and C' and on the free border of the lip just outside D and D'. On the median side of the cleft the whole area included within the lines $C'\ D'\ E'$ is to be removed. On the lateral side the knife is carried from X along the curved line marking the nasolabial junction to C, thence from C to D and from D to E. The whole area of lip included between $X\ C\ D\ E$ is to be removed.

After having planned the whole procedure and fixed the boundaries of the areas to be excised we proceed as follows: The lip on each side of the cleft is separated from the underlying bone over a considerable area, the extent of which is regulated by the mobility of the flaps and the absence of tension when the edges of the cleft are approximated. The ala nasi must be lifted up from its bed so thoroughly that it can swing into position without the slightest tension. The philtrum must be lifted up just as far as the columella, but no further. Bleeding is stopped by sponge pressure. Then the margins of the cleft are steadied by the traction threads and the edges denuded *secundum artem.* We use a strong cataract knife and transfix the lip near the vermilion border at the point D or D', according to the side. We then cut our way along the lines previously marked out into the nostril and on to the free border of lip, removing completely the area of skin and mucous membrane outlined. Thus on the median line of the cleft we excise the area $C'\ D'\ E'$ and on the lateral side the area $X\ C\ D\ E$. Bleeding is stopped by sponge pressure. It is seldom necessary to clamp the bleeding points. We never apply ligatures because it is always wise to avoid embedding catgut or any other foreign material in the wound. The sides of the incision are carefully inspected and if necessary the edges of the skin and mucous membrane are trimmed off with curved scissors. It is usually wise to trim off a strip of mucous membrane to give a broader raw surface of application. Particular attention is given to the pointed flaps $D\ E$ and $D'\ E'$

at the vermilion border. They frequently require considerable trimming before they fit together accurately. Then the points C and C' are approximated, and the ring of the nostril is adjusted and compared with that of the sound side. A little trimming may be necessary, particularly in cases where the triangle $A\ B\ C$ is somewhat indistinct. The sides $C\ D\ E$ and $C'\ D'\ E'$ are then approximated, and if they are found to coincide we are now ready to insert the sutures. If not, they must be trimmed until they fit accurately. The first suture is placed so as to bring C and C' in contact. This can be tied at once. At this stage we usually stitch the nasal mucous membrane along the line $X\ C$ to the raw surface of the nostril inside C'. Sutures are now placed at intervals along the lines $C\ D$ and $C'\ D'$. Great care must be taken in placing the suture at D and D' through skin a little distance from the vermilion border, and not through the mucous membrane; otherwise it will lie in bad holding ground and will pucker up the free margin of the lip when it is tied. The needles must penetrate the skin about $\frac{1}{8}$ inch from its margin, and pass with a slight outward curve *deep into the muscle*, to emerge on the denuded border of the lip just anterior to the line of mucous membrane. When the stitches are tightened the mucous membrane is left free and does not dip between the raw surfaces. The stitches are now tied *seriatim* from the nostril to the vermilion border. Finally the surfaces $D\ E$ and $D'\ E'$ are sutured together by interrupted stitches which pass along the anterior line of contact from D to E at the tip of the flap, and thence upward on the posterior line of contact as high as D in the interior of the mouth. These stitches do not penetrate deeply for fear of strangulation. For suture material we use fine silkworm gut for the skin sutures, horsehair for the exposed mucous membrane of the vermilion border, and fine Chinese silk for the rest of the mucous membrane. We seldom use artificial support to the lip or nostril after the operation, either in the form of button sutures (Blair) or of adhesive strips (Brophy). The stitches are removed on the seventh day after the operation or thereabouts. No dressing except sterile vaseline is used.

The immediate cosmetic results are very pleasing. As time passes, however, there is usually a tendency for the restored nostril to flatten out and for the ala to slip away from the middle

line. This tendency is much greater in cases where the lip is repaired over a gap between the ends of the alveolar borders than it is where the alveolar borders have been approximated at a previous operation. Overcorrection of the deformity of the ala will lessen the subsequent flattening, but this necessitates such extensive undercutting of the ala nasi that we hesitate to employ it in an average case.

Blair has tried to overcome it by extensive separation of the nasal mucous membrane lining the inner side of the ala, followed by shifting of the nasal flap to a higher level in the nostril. The flap is fixed in its new position by a mattress stitch passed from the outer surface of the nose through the alar cartilage, and finally the ala is fastened firmly in the desired position by a button suture. This suture is applied in the following way: It penetrates the skin of the cheek just outside the deformed ala, passes across the anterior nasal space of this side, penetrates the septum and emerges from the sound nostril. Small buttons of lead or tin foil are fastened on each end to hold the suture tight and to support the soft parts when they are placed in proper position. The results obtained by this method are excellent.

Third Stage: Repair of the Posterior Part of the Hard Palate and Velum

This operation, the last of the series, should not be undertaken until the repaired lip has healed completely and united firmly. After the age of nine months, in a well-nourished baby weighing about 20 pounds, with a clean mouth free of decayed teeth, the operation can be done with safety.

At this stage if we examine the roof of the mouth (Fig. 15) we usually find that the anterior ends of the alveolar processes are in contact and united by scar tissue. In some cases, especially those in which the septal flap was long and broad at the time of the first operation, we find that fully two-thirds of the anterior part of the hard palate is closed. The remaining part of the cleft is much narrower than it was at birth, not relatively but actually. The divided halves of the velum and uvula are still wide apart, but owing to the fact that the hard palate on each side in front of the velum, which was moved closer to the middle

line at the first operation, has remained firmly fixed in its new position, the gap is actually much less than it was at birth.

Preliminary Preparation. It is absolutely necessary to have a good view of the posterior part of the mouth and to control the tongue. A suitable gag must be available. There is no such instrument as a universal gag. It is wise to have an assortment ready at hand, because one can never tell beforehand which gag will be most suitable. As a rule, in young children, I use a small "Dann modification of Jennings," with cheek retractors attached. The tongue is controlled by a silkworm-gut stitch passed through its tip.

Steps of the Operation. A traction stitch of fine silk is passed through the fleshy part of each velum about $\frac{1}{8}$ inch from its free edge. It is used exclusively as a tractor. If it is handled gently it does not bruise or tear the tissues and enables us to manipulate the velum more easily and quickly than by hooks or forceps. An incision (Langenbeck's) is now made through the mucoperiosteum of the hard palate along the alveolar margin on one side (Fig. 16, *C* and *D*). It extends from a point well in front of the anterior end of the cleft to the maxillary tuberosity. Thence it courses around the tuberosity to its outer border, and thence for a short distance along the prominent ridge of mucous membrane, stretching from the tuberosity of the maxilla to that of the mandible. The incision is carried to bone, except along the line joining the tuberosities. Here, at first, it penetrates mucous membrane only. By means of periosteal elevators the mucoperiosteal layer is lifted up from the bony palatal plates as far inward as the edge of the cleft and as far back as the posterior border of the palate bone. The velum is now lifted upward by the traction suture, and the palatal aponeurosis and nasal mucous membrane are divided along the line of the posterior border of the palate bone by careful snips with curved scissors. The whole palatal flap, consisting of mucoperiosteum and velum, can now be lifted upward. The anterior end of the flap is formed by a narrow mucoperiosteal strip, which is continuous in front with the undisturbed mucoperiosteum which covers the front of the palate. It is covered by mucous membrane on its oral surface, but raw on its cranial. The posterior end of the flap consists of the whole thickness of the velum. It is covered by mucous membrane on both its

cranial and oral surfaces. So far the vascular supply of this flap has not been hurt. Blood can reach it from its anterior connection through the nasopalatine artery, and from the velum through the greater palatine artery. In suitable cases, with a narrow cleft, the edges of the flap can be brought together at this stage without tension, and consequently, further liberation of the flap is unnecessary. Generally, however, the cleft is too wide and the tension on the flaps too great to justify suture without further liberation. We then proceed as follows: By the vigorous use of a periosteal elevator, inserted into the curved incision carried around the maxillary tuberosity, we peel up the mucoperiosteal flap from the underlying bone and force it bodily inward toward the hamular process. With care there is no danger of wounding the greater palatine artery. The whole velum can in this manner be forced inward for a considerable distance and held in its new position by a gauze pack inserted tightly into the incision. If necessary we can go a step further. By cutting the pterygomandibular raphe (Fig. 10, *B*) along the line of the original incision in the mucous membrane which covers it (*vide ante*), we can sever the connection between the buccinator muscle in front and the superior constrictor of the pharynx behind. Such an incision will allow the whole velum, together with the faucial pillars and tonsil, to be shifted toward the median line without endangering the arteries reaching the velum from below.

Although we employ these extensions of the lateral incision in unusually difficult cases, we prefer, as a rule, to relieve tension by the simple expedient of dividing the pharyngopalatinus and levator veli palatini muscles. They can be cut across safely in the following manner (Figs. 18 and 19): If the soft palate and uvula be lifted up by the traction stitch both pharyngopalatinus and levator veli palatini can be brought into view. They both stand out in strong relief—the one as a thin, narrow, muscular band just behind the tonsil; the other as a rounded, firm, muscular bundle situated deep on the lateral wall of the pharynx just behind the open mouth of the auditory tube. They are divided with curved scissors, the pharyngopalatinus at a point just behind the middle of the tonsil and the levator veli palatini just behind the opening of the auditory tube.

The degree of relaxation obtained by the division of these

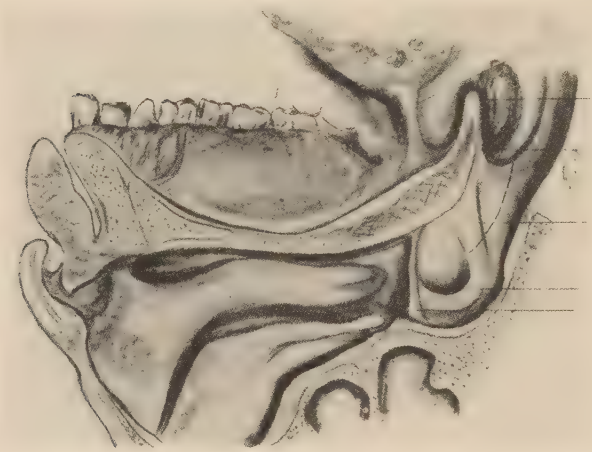


FIG. 18.—Sagittal section through the nasal and buccal cavities showing a lateral view of the nasopharynx. The mucous membrane covering the levator palati and palatopharyngeus muscles has been undisturbed. These muscles produce prominent ridges. The labeling is descriptive. The specimen has been placed in the same position as the head of a patient during the actual operation. The dashed curved line shows the position of the incision through the mucous membrane when the palatopharyngeus and levator palati muscles are divided to relieve tension.

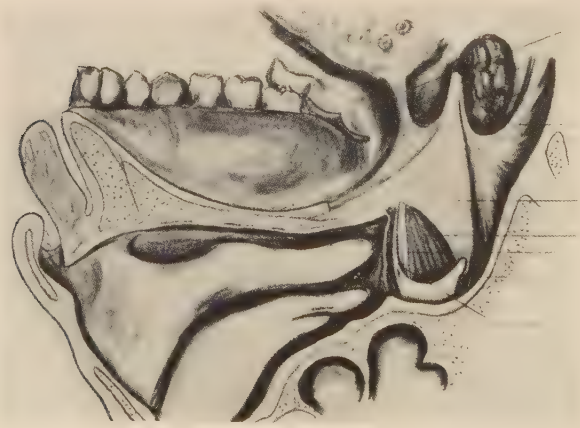


FIG. 19.—The same specimen from which Fig. 18 was drawn, dissected to show the muscles passing into the palate from above and behind. The large bundle representing the levator palati is very clearly shown. Note how accessible it is. The labeling is explanatory.

muscles is astounding. Hemorrhage is slight and easily controlled by sponge pressure. The muscles unite promptly and we have never seen paralysis and atrophy of the muscles follow the procedure.

The same steps are repeated on the opposite side of the cleft.

Paring of the Edges of the Flaps. In the hard palate this is unnecessary if mattress sutures are used; in the soft palate it must be done thoroughly and with the greatest care. The mucous membrane must be removed in a single, complete strip to avoid leaving small islands behind. On each side the soft palate is transfixed by a cataract knife, and a thin strip of mucous membrane is removed from its free edge, extending from the tip of the uvula as far forward as the junction of soft and hard palate.

Insertion of the Sutures. It is often convenient to secure control of each side of the palate by means of traction stitches. Usually we insert three on each side—one in front, another at the junction of hard and soft palates and a third through the thickness of soft palate lateral to the uvula. Blair prefers to handle the palate by means of a tenaculum, made by fixing the broken point of a sewing needle in the jaws of a pair of forci-pressure forceps. The first suture is passed through both edges of the cleft at the junction of hard and soft palates. This is not tied, but is merely used to lift up the flaps and to secure accurate spacing of the rest of the sutures. The sutures are now passed systematically from before backward. Each stitch is of the mattress type, inserted in such a manner as to avoid strangulating the blood supply to the edges of the flap (vertical mattress stitch, Fig. 20). Three or four stitches may be required in the hard palate. When tied the raw (upper or cranial) surfaces of the flaps are opposed to one another and the edges of the flaps are turned out like the flange in a steam-pipe. Similar stitches are passed in the velum, care being taken not to penetrate through the mucous membrane on its cranial surface. The stitches in the uvula must be passed very carefully, so as to avoid strangulation when they are tied. Mattress stitches are unsuitable. The ordinary through-and-through variety, passing through mucous membrane and penetrating about half the thickness of the uvula, serve the purpose best. When all the sutures are tied, the uvula is pulled forward and several sutures are passed through the edges of the mucous membrane on its cranial

surface. By proceeding systematically from before backward, and using the last stitch passed as a tractor to hold up the flaps while the next stitch is inserted, little trouble is experienced in placing the sutures in good position. The suture material varies: For mattress stitches we use carefully selected, fine silkworm gut; for the other stitches fine silk.

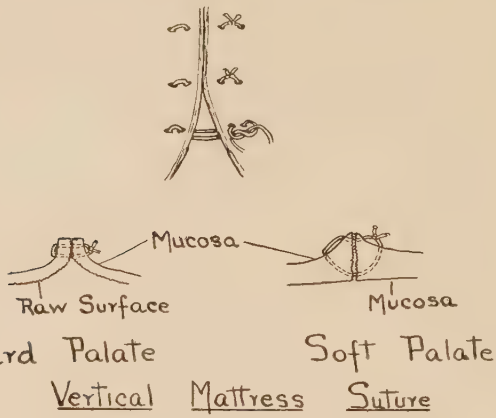


FIG. 20.—Shows the method of passing the vertical mattress sutures. In the upper figure the two upper stitches are tied very loosely; the lower one is not tied. It is clear that stitches passed in this manner cannot strangulate the blood supply to the edges of the flaps. In the lower figures, we see cross-sections of the stitches tied tight. On the left, the raw surfaces of the hard palate are seen to be held firmly together over a large area, and the edges turned out like the flanges of a steam pipe. On the right, the stitch is shown passing through mucosa on buccal surface of palate. but not penetrating mucosa on cranial surface. This technic is of the greatest value,

For several years we have abandoned the use of metal buttons or plates to prevent undue tension on the line of sutures. We found that they were both irritating and dirty, and that they could be dispensed with if proper relaxation was secured by other means.

We seldom use gauze packs in the lateral incisions, for the same reasons, except when operating on the palates of adults, in whom postoperative bleeding is not at all uncommon. In these patients we pack the lateral incisions tightly with gauze soaked in collargol. It serves a double end; it reduces tension on the sutures and stops bleeding from the raw surfaces. The pack should be removed forty-eight hours after the operation.

After-treatment. The traction stitch is left in the tongue until the baby has completely recovered from the shock of the operation and respiration is tranquil.

Plenty of water is given by mouth from the start. We never use any medication or take particular trouble to inspect the field of operation. An offensive odor to the breath and continued fever, lasting beyond the second day of convalescence, are ominous signs pointing to wound infection. We seldom remove any of the stitches unless they are retained longer than two months. Usually they come away spontaneously.

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DISCUSSION ON THE PAPERS OF DRS. DAVIS, HORSLEY, DOWD, RITCHIE, SISTRUNK, McWILLIAMS AND LEWIS, SHIPLEY, LYLE AND THOMPSON

Dr. Allen B. Kanavel, Chicago, Ill.:

There are to me very many interesting points in this discussion of plastic surgery. One is the question of transplanting the full-thickness skin-graft in the cheek. I have had the same difficulty in transplanting full-thickness grafts on the cheek. I have met the problem by taking ordinary modeling compound, laying it on the inside of the cheek and having the patient bite down on it so as to make the teeth fit into it. By leaving the teeth open for about ½ inch the patient can be fed through the mouth. With the flat, hard compound supporting the buccal surface, the graft can be laid on the cheek and pressure maintained over the graft with a satisfactory result.

In the matter of transplanting skin in the palm, Dr. McWilliams spoke about the difficulty of transplanting skin by pedicles and having a pad of fat left. I have too many of those cases, done in earlier years, with a large pad of fat in the palm. In the last half dozen years I am sure that my pedicled flaps in which I have cut off practically all the fat have been just as satisfactory as have been those in which I have used a free, full-thickness graft. I do not place a full-thickness, free graft on the tendons in the palm because a disability may result from contracture of the tendons. I have used free grafts, however, very satisfactorily in webbed fingers. In these cases I have cut the web and covered the raw surface with full-thickness grafts. There they are much more satisfactory than Thiersch grafts.

There are two other problems I would like to speak of for a moment. One is that of filling the orbital cavity after the removal of the orbital contents and lining periosteum in such a way as to permit the patient to wear an artificial eye. In these patients I have transplanted a pedicled flap of fat at the primary operation so as to fill the orbit with living tissue. At the end of two weeks I have divided the pedicle, leaving the fat growing in the orbit. After two or three weeks I have taken an oversized glass eye, wrapped a Thiersch graft around it and sewed the eyelids down over the graft. In 2 cases I have been able to secure a completely epithelial-lined cavity in which an eye could be placed.

The other problem is that of repairing a defect due to malignant growth or syphilis, in which there is a destruction of the skin of the forehead and an opening into the frontal sinus. In 3 cases I used a pedicled flap of skin, sufficient fat attached to it entirely to fill the

frontal sinus, and in all 3 cases I secured a satisfactory transplant, both as regards the fatty portions of the flap and the covering skin.

Dr. Charles H. Mayo, Rochester, Minn.:

I too have been full of that spirit of adventure and expectation that is really an attribute of the plastic surgeon and sometimes also his solace.

This morning the matter of the bare bone of the skull was discussed. I have seen other places where the bone was bare and dry. Years ago I suggested taking a little drill and drilling down into the diploe, until it bleeds, every $\frac{1}{4}$ inch all over that area of the skull. It takes only ten or twelve days for little buttons of granulation to come out; as they approach each other they will tolerate almost any kind of plastic graft. It is a very easy thing to do.

It is not loss of blood that kills a flap; when a pediculated flap is put under tension it is at first too white; if it is going to die it will later turn dark with more blood than it can get rid of. If, just before the flap is sutured, a great many punctures are made all over it with a sharp knife, these punctures will serve the place of veins for forty-eight hours and will allow the accumulation of blood and serum to leak out at each point of incision. It is a very useful procedure on some of those flaps that you throw in covering breast areas following operation for cancer.

In the repair of harelips and cleft palates, especially the facial part of the operation, it is well to loosen everything up and cut underneath the good side to prevent it from continuing for the next six months to get the best of the bad side. Growth of the good side should be reduced by cutting underneath next to the bone just far enough to give them both the same chance. If this area of the face is loosened up it will reshape itself. It may seem like a poor proposition to cut underneath the good side. It is not. It will leave it perfectly loose and it will adjust itself to the middle line instead of growing better and distorting the abnormal side.

The modern pathologist classifies the grade of malignancy as 1, 2, 3 or 4. A carcinoma graded 4 is so embryonic in type of cell that it is difficult to distinguish it from sarcoma; but we do not see a patient with carcinoma graded 4 get well and stay well. The pathologist should commit himself concerning the virulency of the cancer so that you can mark down, "This is a case of cancer in which the cancer cells resemble those of the tissue from which they came; this case would be suitable to work at a second and a third time. If it recurs it will be local and not metastatic." That will be of great aid when patients who live at a distance report that they are having some trouble. To them you can say, "Come back; more can be done for you. You had the most benign form of cancer."

Dr. Emmet Rixford, San Francisco, Calif.:

Plastic surgery is the highest form of the art of robbing Peter to pay Paul, speaking in a surgical sense. There is one principle of very wide application that has not been mentioned today which I think is worthy of being mentioned very briefly, and that is that employed in Langenbeck's cheiloplasty.

It simply is the taking of two flaps with opposite pedicles, lifting them up and reversing them. The idea is that in that way one can steal from the surrounding tissues in all directions and cover the defect, with a minimum of tension, a minimum of puckering and a minimum of loss of tissue. The resulting scar is a simple letter S or Z. We call it Langenbeck's cheiloplasty because it was on the lip that Langenbeck used it. I suppose it really was invented far earlier, possibly by Hippocrates, but it is continually being rediscovered. Our friend, Dr. Jackson, rediscovered it and put it on the chest, in the so-called Jackson flap, in closing the wound after removal of the breast for carcinoma.

The Frenchman who treated syndactylia by flaps on opposite sides did the same thing in three dimensions. We can do the same thing in three dimensions for all webs; for example, in the web of the thumb, simply splitting the flaps parallel to the skin surfaces, cutting down on one side and in the opposite direction on the other side, which makes two flaps with opposite pedicles, then reversing the flaps, the skin comes together and the wound is closed. Really the thing is very beautiful. It is a principle of the very widest application in plastic surgery.

In the matter of removing mediastinal and costal metastases, making and covering large defects in the chest wall, I am pleased to hear Dr. Shipley's paper and to have other judgment that it is really worth while, because at the meeting of the American Surgical Association in San Francisco, in 1905, I had the honor to present a number of cases of very similar character, and the general burden of the discussion then was that it was not worth while.

Dr. Mayo has given the secret of the thing, that in those cases of carcinoma in which there is a minimum of virulence one can sometimes, by repeated operation, greatly prolong life and sometimes cure. In one of the cases that I had the pleasure of reporting on that occasion we had a recurrence along the internal mammary artery. We opened the chest wall and took out that and half the sternum without any trouble; then subsequently there was a recurrence on the other side, so we opened the chest on the other side and took out the rest of the sternum. After the second operation the patient was well for some six years, dying, the physicians said, of apoplexy. I suspect more likely it was a cerebral recurrence. At any rate she had six years of normal health.

In the matter of opening the chest wall under such circumstances it is not necessary to use differential pressure if one opens only the one side, but it is important to let the air in slowly at first. If one will convert the open pneumothorax into a closed pneumothorax, by sliding a wet towel underneath the flap as you open it and making some resistance, it need not be complete resistance, to the ingress and egress of air to and from the chest, one can maintain a fairly normal respiration without great risk.

Dr. Vilray Papin Blair, St. Louis, Mo.:

In attempting to discuss these papers one is in about the same fix as the hungry small boy turned loose in a candy shop with only two minutes.

I think Dr. Davis has given us a very concise rounding up of this subject. There is just one point on which I would disagree with him in his presentation. I think he will agree with me. He spoke despairingly about decorative surgery. We cannot overlook the fact that the consciousness of a real or a fancied defect will ruin a person's comfort, usefulness and happiness in life, just as much as a stomach ulcer. It takes a great deal of study, sometimes, to figure just what the proportion is between the physical defect and the mental attitude, but if one throws these aside one is going to miss a large part of the calling. I am not referring to the vain woman who simply wants something that is of questionable value.

Ninety per cent of our work in that type of case is either with working people or the families of working people. Whether it is going to help a woman who is getting old, who is supporting herself or a family, to hold her position; whether it is going to give confidence to a man or a boy to put over his position; or whether it is going to help a woman hold her husband—the work is just as useful and just as important as the curing of any bodily disease. It is the hardest type of surgery to handle, but you cannot dismiss it.

I think Dr. Horsley's paper is a classic and that he has put in categorical form certain questions which confront all of us. I am still waiting for him to give me the answer.

Dr. Ritchie with his slides has proved his point, but when he wrote to me that he wanted me to test out these lips with a battery, with all the other complications I had had trying to figure the thing out, I felt very much like Dr. Seelig's young Jew, who, injured in front of a church and covered with blood, was asked by the minister if he believed in the immortality of the soul, said, "Me in this fix and they ask me conundrums yet."

I do not think anybody is ever going to seriously disagree with Dr.

Thompson on any point of surgery that he puts up, but I certainly disagree with his illustrations. You owe to Dr. Thompson, as far as I know, the tribute of being the first one to introduce a pair of calipers for accurately measuring the lip, just as a tailor might use a ruler or a tapeline to measure cloth before he cuts into it. It seems a long time for the world to have gone on guessing before he came along.

Dr. Sistrunk's presentation is characteristically ingenious. If he has never seen that general plan of pulling the edges of the skin together, and piecemeal taking out the scars referred to, he will find it touched upon at least in a review of Eitner's work published in *Surgery, Gynecology and Obstetrics* a few years ago.

Dr. Dowd has proved the point that there are many roads to Rome. The most important thing about the road is that the man who travels it knows the particular road and sticks to it. Nothing could be prettier than the result he showed you.

This surgery is usually a practice of robbing Peter to pay Paul, but many people who have a deformity of any kind on the face or neck are going to be much more sensitive to putting another scar in the neighborhood than a person who has not that kind of deformity. They have a mental impression of that thing, and the older ones (not the child), hate to have any more cutting around that area. For that reason, if for no other, there are certain advantages in getting your material from a distance where the new defect will not show; but if you do this you are going to have a different-colored material.

There were two or three of Dr. McWilliams' points with which I did not quite agree. One I disagreed with was that he said homografts are uncertain in result. Our experience is that they are perfectly certain—all bad. Others seem not to have had this experience.

Dr. Frank L. Hupp, Wheeling, W. Va.:

Arm, chest or brachiothoracic adhesions following serious or neglected burns constitute one of the very important problems of the plastic surgeon. Too often these little children are almost hopelessly burned, perhaps a third of their body involved, and their agony is so great that it is almost impossible to abduct or extend the arm at the time of the accident. If they recover, the arm is not only helpless as to function by reason of the contracture, but growth is retarded and there is marked atrophy of the humerus.

In his masterpiece on this subject which appeared, I believe, in the *Archives of Surgery* (January, 1924) Dr. Davis has impressed us with the importance of exercising charity and refraining from criticism when these cases come to us for operation. With Dr. Davis, I feel that the key to the situation is successful reconstruction or reformation of the

space in the axilla. Every constricting band or adhesion should be carefully cut up to and including fibers of the muscles, using a cushion of fluffed gauze or a sea-sponge to support the flap beneath the axilla. The operative measure must be done in steps or it becomes an extra-hazardous risk.

FIG. 1.—Showing arm-chest or brachio-thoracic adhesions following extensive burns.

FIG. 2.—Posterior view.

I should like to emphasize two other points: first, to make allowance for the shrinkage of the flap in covering the denudation of the arm and on this point I just take issue with Dr. McWilliams. He said this morning that the flap must be the exact size of the area to be covered. I think he refers to whole-thickness transplants. But certainly in these axillary pads they must be cut much in excess of the space to be covered. The second point is to provide against the

Fig. 3.—Aeroplane splint.

Fig. 4.—Reconstruction of axilla with full-thickness skin transplant from chest.

cicatricial circulatory interference in transposing the flap to the arm. In one instance I recall a contracture after a very satisfactory piece of surgery had been performed where the circulation was temporarily, at least, interfered with to such an extent that there was some slight ulceration of the tips of the fingers, much the same as one would see in a thromboangiitis obliterans.

FIG. 5.—Left picture showing *x*-ray of humerus: note marked atrophy from disuse.

We have to remember, as Dr. Davis has told us, that the object of the operation is to restore function, to relieve deformity, to prevent recurrence, and the complete restoration of the axilla.

These slides illustrate a typical case of severe burn with the resulting brachiothoracic adhesions and extensive cicatrization. One of the slides, which is a reproduction of an *x*-ray picture, visualizes in a striking way the marked atrophy of the humerus by reason of long disuse and disturbed circulation.

The axillary reconstruction is well illustrated; and the functional result all that could be desired.

DR. CHARLES L. GIBSON, New York City:

I just want to remind the members of the Association of a procedure of which they all know and probably have practised, but which most of

us have forgotten. It has been rehabilitated very skilfully by Dr. Beck, of Chicago. I mean the principle of inducing epithelium to grow from the sound edges over a cavity. Given a perfectly good, clean, healthy, granulated surface, this method is sure and gives a better-looking skin eventually than any other method. It is sure in that the flap does not slough or anything of that sort.

If you remember the article by Dr. Beck, one-half goes on the sound skin, one-half on the other surface. If you take that off, the next day you find a little, thin, bluish film. The day after it is advanced. It is astonishing that in about a week the large cavity has been reduced one-half, and it takes, for the average cavity—for instance, the palm of the hand—about three weeks to cover that with perfectly good, sound skin, skin that is always going to look and act just as good as it did when it grew there originally.

I should like to call attention to a paper which Dr. Walther, of Paris, intended to read when he was invited to be a guest here, I think in 1890, on the treatment of elephantiasis. It is so simple that I think it ought to be mentioned. We have been told this morning that these operations have considerable shock and that they seem formidable. I have seen some of Walther's patients and they are far the best results I have ever seen. The thing is simplicity itself. Dr. Walther introduces a piece of rubber tube and carries it superficially to the fascia under the skin. This groove dips down to establish communication. That is all there is to it. The tube is sewed in place, is left in there and it gives a perfectly good result.

Dr. Evarts A. Graham, St. Louis:

I should like to say just a word about the question of pressure brought up by Dr. McWilliams this morning, illustrating the use of the small rubber bag which he passed around. About six years ago Dr. Blair undertook some experiments on the effect of pressure in relation to the healing of large flaps, using much larger appliances than this small rubber bag. After experimenting with various artificial mechanisms, he introduced the marine sponge as a dressing (I believe he was the first to introduce it), which after some experience one can regulate perfectly, constituting the proper degree of pressure to use on these large flaps.

It seemed to me that a small rubber bag, such as was passed around this morning, could be of very little service in a large flap. Certainly something larger is necessary than that small rubber bag

I might say also that the sea-sponge which I have just described is an exceedingly useful procedure for obliterating the subclavicular space after radical operations on the breast. It is applied on the skin flap

and kept in position for two or three days, thus preventing the collection of serum beneath the flap.

Dr. John H. Gibbon, Philadelphia:

It is a well-recognized fact that the principal thing in defects of the chest wall is the closure of the pleura immediately. Those of us who had to deal with gunshot wounds of the chest during the war tried many methods of closing the pleura. Where tissue was lost it was impossible in many instances. I tried a number of times to utilize a method which failed, but which in civil life has succeeded at least once. It is so simple that I thought it worth mentioning.

Ten years after the removal of a breast a patient presented herself with a local recurrence. In the excision of it it was necessary to remove two or three ribs and the underlying pleura, leaving quite a large defect in the wall, with collapse of the lung and the pericardium in plain view. The area was nearly as large as the palm of a hand. The patient was an old woman and not in good condition. Instead of attempting a plastic, I tried once more what I attempted in the war. I took a very thick, large sponge, thoroughly impregnated with sterile vaseline, tucked it under the skin edges, fixed it there, put on a large dressing with enough pressure to cut off the ingress and egress of air and left this dressing on for two weeks. No infection occurred. When the vaselined gauze was taken off gradually, a few layers each day, the pericardium and pleura were adherent and covered with healthy granulation. In a few weeks, with exposure to air, the area entirely healed. I think you will find it is a very good method if you have a sterile wound.

Dr. Davis (in closing): The point spoken of by Dr. Blair, concerning certain cases for which something has to be done in the way of cosmetic surgery, was considered in my longer paper.

Two important points were brought out by Dr. Dowd: One was the use of scar-infiltrated flaps. While these are not as good as normal tissue, I have found in many instances that tissue infiltrated with scar can be shifted very satisfactorily. The other point was letting time elapse after the insertion of the flap before finally straightening it out; let the patient go home and return in six months and then straighten out the remaining kinks.

As to Dr. Ritchie's muscle theory: He wrote me about it some time ago, and I have tried it on possibly a dozen cases and have found it extremely useful. I did not know how to use the battery, just as he showed us this morning, but I was able to check up on the approximation of the muscle ends and found that it was very helpful.

Dr. Sistrunk's remarks on the removal of scars in stages calls attention to an old procedure and a very useful one. I have used it for years on *x*-ray burns, of the type you can slide, also when dealing with keloids, big angiomata, etc. Morestin, in France, wrote an interesting paper on the same subject about 1916.

I feel very much about the dressing of whole-thickness grafts as does Dr. Graham. The sea-sponge, first brought out for this purpose by Dr. Blair, has been very satisfactory in my hands, and personally I see no particular reason to change to rubber bags.

Dr. Horsley (in closing):

While the histologic examination of tissue of a cicatricial contraction gives no direct positive evidence as to the cause of the contraction, the negative evidence is interesting. Observations of other phenomena and histologic examinations of other tissues, which have been described in the body of the paper, throw considerable light upon the causes of cicatricial contraction.

The quantity of scar tissue, as discussed under the heading 2-f, probably has much to do with cicatricial contraction, though in many instances extensive scar tissue is not followed by marked contraction. This is when keloid or pseudo-keloid formation takes place and the scar tissue seems to have abundant nourishment, or for some other reason individual cells and fibers of the scar tissue do not shrivel, which seems to be essential for cicatricial contraction.

Dr. Dowd (in closing):

Dr. Davis has already called attention to the points I wished to bring out.

Dr. Ritchie (in closing):

I hope that I have not added any confusion to the subject. What I wanted to develop, at least for my own satisfaction, was a more definite purpose, a more definite motive with which I could approach every case in the repair of the lip. How far it goes in the cosmetic repair of the ala and the nostril I am still uncertain.

There are three operations in the literature of the lip that very nearly meet the muscle theory: the old Mirault operation, which has an undercutting of the ala; the Rose operation, that prepares for lateral approximation, but I believe sacrifices too much tissue; and the most acceptable and intelligible plan in modern literature, Dr. Thomson's caliper operation, which meets most nearly the end-to-end anastomosis of this muscle.

What I see in the muscle theory is that we change over from the indirect special methods of harelip operation to the direct steps of general surgery.

Dr. Thompson (in closing):

I felt when I presented my paper that I really wanted, if possible, to place the operation on an anatomic basis. I think that we have been doing a great many plastic operations without an anatomic basis and getting results without knowing why. I think anything that adds to an intelligent understanding of the procedure is worth while.

A little time ago I talked to Dr. Ritchie about his operation, and he was kind enough to send me a battery. When Dr. Ritchie came to visit me he demonstrated the muscular contraction of the orbicularis muscle in a typical case of the cleft lip. It is really very beautiful when you see it. I found that I had been making my cuts well out into muscle without knowing it, and that I was getting much better lips than I used to get in times gone by, before I undercut the ala so much. That pleased me, because I always like to understand things.

Dr. Ritchie also told me that some doubt had been raised as to whether the lip covering the premaxilla contained muscle or not. While Dr. Ritchie was with me I operated upon a case of bilateral cleft of the lip, complete into the nostril on one side but incomplete on the other side. Here the lower margin of the nostril was closed by a narrow bar of tissue, which is the remains of the lateral nasal process about which I spoke to you this morning. We stimulate the lip covering the premaxilla and produced mild contraction. We found histologically it had muscle. The orbicularis oris muscle probably reaches the covering of the premaxilla by migration of the muscles covering the maxillary processes. We see an analogous condition of migration in the platysma, which is originally formed in connection with the hyoid arch, which is supplied by the seventh cranial nerve. All the muscles of the face supplied by the seventh nerve probably migrate in the same way and carry their nerves with them.

These observations are of intense interest. They open up a delightful vista, which is the romance of surgery.

Dr. Sistrunk (in closing):

I am sorry Dr. Gibson did not understand the type of case to which I referred in the operation suggested for lacerated anal sphincter. I tried to make it clear that the type of case for which this operation was suggested was one in which several operations had been previously performed. I think we often see a case in which considerable sloughing of the tissues has occurred following a large perineal abscess or the

muscle has been practically destroyed by repeated operations. Very good results may be obtained from the operation usually performed in simple tiers when no previous operation has been attempted.

I was also interested in the question which was raised in regard to treating elephantiasis by introducing a rubber tube into the subcutaneous tissues. This procedure makes one think, of course, of the use of the silk strands suggested by Handley some years ago. I think there is no question that temporary relief follows an operation of that sort. I operated on one woman for the removal of several large silk threads which had been placed from the ankle up to the inguinal region and on to the abdominal wall several years previously. Sections made from the tissues surrounding these silk strands showed that the silk had become surrounded by very dense fibrous tissue which prevented its acting in the way which Handley had thought it might; that is, by capillary attraction.

I operated on another patient with elephantiasis in whom a rubber tube had been buried on each side of the limb about two years previously. The tubes were also surrounded by fibrous tissue. My own opinion is that such operations give temporary relief only.

Dr. McWilliams (in closing):

Dr. Hupp and I were talking about two entirely different things. Dr. Hupp was talking about pedicle grafts and I about free full-thickness grafts. Free full-thickness grafts must be no larger than the size of the defect, but pedicle grafts must be at least one-third larger. As for the use of the sponge *versus* the rubber bag, I think the rubber bag makes the thing foolproof. Thiersch grafts are very easy to make and are foolproof, so to speak.

In the first three or four grafts that I made of free *full-thickness* grafts I lost the graft. I discovered it was because I did not put enough pressure on. If you use these bags with 30 mm. of pressure, it has a proper degree of pressure. They are made in different sizes. They can be made by Victor Mueller, of Chicago, in any size you like. Those who are not as skilful as Dr. Davis and others are, by experience, in putting the proper degree of pressure upon these grafts, I think, with this method of accurate pressure when they are beginning, will find the full-thickness grafts will take the place of the Thiersch grafts.

ALBERT JOHN OCHSNER, M.D.

REPORT OF COMMITTEE ON ARCHIVES

ALBERT JOHN OCHSNER, B.S., M.D., F.R.M.S. (Eng.), LL.D., F.A.C.S., Hon. F.R.M.S. (Ire.)

1858–1925

Albert John Ochsner was born, April 3, 1858, at Baraboo, Wisconsin. His parents were Henry Ochsner and Judithea Hottinger. Henry Ochsner, with his brothers, Conrad and John, left Nanikon, Canton Zurich, Switzerland, in the forties to make a new home in the new world. The Ochsner family could trace its lineage back to the time of Paracelsus, whose mother was Anna Ochsner. For many years they had played an important rôle in the life of the "gemeinde."

Albert John was the second child, and soon after his birth the family moved to a large, fertile farm in Honey Creek. Here he spent his early life and procured a rural school education. When this was completed he went to Baraboo to attend the high school. Following this, he spent several years in teaching, and in 1881 entered the University of Wisconsin. In three years he finished the four-year course and was first man in his class. In 1886 he was graduated from Rush Medical College, and then spent the next three years in graduate study abroad. For several years he was associated with and influenced by such men as Gunn, Parkes, Senn and Fenger. In 1891 he became chief surgeon to the Augustana Hospital, which at that time was a small hospital of thirty beds. Due to his ability and tireless energy, more room was needed and soon a new hospital was built. Additions from time to time have been necessary, and at the time of his death he was busy building a new hospital with twice the present capacity of the old one.

Always tireless, with an energy that surmounted all obstacles, he early attracted the attention of his colleagues. Unassuming, he seemed to radiate a presence that made itself felt. He always commanded respect and was always respectful of the views and opinions of others. Known the world over, his name or his letter served as an introduction in any clinic. His writings, which were many, were noted for their simplicity, brevity and ability to make a lasting impression. His clinics were always filled with visitors and all the procedures were kept as simple as possible, that men coming to the clinic might learn and be able to use his methods. He was never too busy to lend a helping hand to a colleague in trouble or to a person who needed his expert services. He was one of a very few men who possessed instinctive clinical judgment and clinical ability in diagnosis.

During the early part of his surgical career he planned his work to enable him to spend a certain time every few months visiting the clinics of this country. This he continued and later included the clinics of Great Britain and Europe. He had planned to attend the International Congress of Surgeons to be held this year in Rome. He was a familiar figure at the local meetings, never missing one if it were possible to be present and never failing to call the attention of his associates to the fact that there was a meeting to be attended.

He was known affectionately and respectfully as "the professor." In nearly every city in this country there are medical men who have felt the magic touch of his personality. The career of many a student has been guided and developed by his inspiration.

After a life full to the uttermost, he has gone as he would have chosen. Stricken on Tuesday, July 22, 1925, as he was preparing to go for his daily ride, he rallied sufficiently to perform several operations Thursday morning. Friday afternoon his condition became worse, and on Saturday, July 25, 1925, the great soul passed on. The beacon light that shone so brightly for so many years is gone. Through the reflected light of the many lives he touched his great spirit lives on.

E. R. S.

SAMUEL J. MIXTER, M.D.

SAMUEL J. MIXTER, S.B., M.D., F.A.C.S.

1855–1926

In the death of Samuel J. Mixter the American Surgical Association has lost not only one of its most devoted and active members, whose presence at our meetings was looked forward to by us all, but one who, in the constant companionship of his delightful wife, was the center of some of the most charming memories of our annual gatherings.

Born on a farm in Hardwick, Massachusetts, on May 15, 1855, of an old New England family, he imbibed from his early associations a love of Nature and of the out-of-doors that lasted throughout his life, and in the indulgence of which he found his greatest rest and recreation. Hunting and fishing were his favorite diversions, and during his later years he loved especially to go back to the old homestead in Hardwick, where he and Mrs. Mixter dispensed a hospitality which will never be forgotten by those who were privileged to enjoy it.

From a school in Amherst, Massachusetts, he entered the Massachusetts Institute of Technology, graduating in 1875 with the degree of S.B. Then came the Harvard Medical School, from which he received his degree in 1879, after serving a year as house surgeon at the Massachusetts General Hospital. This was followed by a year of study abroad, and in 1880 he returned to Boston and began the practice of his profession.

Of a naturally mechanical turn of mind, he was from the outset particularly interested in anatomy and surgery, and to them practically the whole of his professional life was devoted. He associated himself at once with the anatomical department of the Harvard Medical School, was appointed assistant demonstrator of anatomy in 1882 and demonstrator in 1887, serving in the latter capacity until 1893. His work in this department was particularly conspicuous for his beautiful corrosion preparations of the internal organs, using for his injection material an alloy that fused

at a comparatively low temperature. These, with many of his anatomical preparations, are preserved in the Warren Museum and attest the interest, ingenuity and thoroughness that was characteristic of all his work.

He was instructor in surgery at Harvard for many years, assistant surgeon at the Carney Hospital from 1880 to 1897, surgeon to out-patients at the Massachusetts General Hospital from 1886 to 1894, and visiting surgeon from 1894 to 1915, at which time he retired to become a member of the consulting staff, a position which he held at the time of his death.

During all this time he was engaged in a very large and active surgical practice, covering a great part of New England, and was consulting surgeon to many of its smaller hospitals.

He was particularly interested in the surgery of the esophagus, Gasserian ganglion and the gall-bladder, to all of which he made valuable contributions.

The broad extent of his interest and experience is attested by the numerous addresses before medical societies and his contributions to medical journals. One of his most important addresses was that as president before this Association in 1917 on "Conservatism in Surgery."

A bold operator, where boldness was necessary, brilliant always in the technic of his dissections, he had a horror of unnecessary mutilation and was ever actuated by a very high ideal of the responsibility of the surgeon to his patient.

He was one of the first to be commissioned lieutenant in the Medical Reserve Corps of the United States Army, was called to duty early in 1917, served as an advisor to the Surgeon-General's Office in Washington, and as inspector and consultant in various camps, retiring as lieutenant-colonel at the close of the World War and was a colonel in the Reserve Corps at the time of his death.

Dr. Mixter was always much interested in the affairs of the Massachusetts Institute of Technology, was president of the Alumni Association in 1904 and a member of the Corporation from 1916 to 1921.

He was a member of the American Academy of Arts and Sciences, of the Southern Surgical Society, a founder of the New England Society and its first president, as well as a member of numerous other professional and social organizations.

He became a member of the American Surgical Association in 1893, was elected president in 1916 and had the great satisfaction of seeing two of his sons honored by election to its fellowship.

His long and distinguished career won for him a very high place in the history of New England surgery. "The chief," as he was affectionately referred to by all of his house officers and assistants, had a place in their hearts that it is given to but few men to obtain. Always a most loyal and devoted friend, he was never so happy as when he could gather a group of his friends about him and dispense a hospitality and cheer that can never be forgotten.

His home life, into which his friends were always more than welcome, was unusually beautiful, and the sudden death of Mrs. Mixter on January 30, 1925, brought to a close more than fifty years of ideal companionship. From the shock of her death Dr. Mixter never fully recovered. While on a visit to a friend's house at Grand Junction, Tennessee, he caught a severe cold, resulting in pneumonia, and died on January 19, 1926.

The happy, cheerful, ever-friendly atmosphere in which they lived, and which they always spread about them, will be a cherished memory as long as any of us who were privileged to come in contact with it shall remain.

H. G.

HENRY R. WHARTON, M.D.

HENRY R. WHARTON

1852–1925

Dr. Henry Redwood Wharton was born in Philadelphia, May 23, 1852. On his father's side he was descended from a line of English and Welsh Quakers. The Whartons have been a very prominent and influential family in the city of Philadelphia and in the Commonwealth since the days of William Penn, and Dr. Wharton's ancestors also included members of the Carpenter and Hollingsworth families, notable Quaker stock. On his mother's side he was of Scotch-Irish extraction, and by virtue of descent a member of the Sons of the Revolution.

He was educated in private schools in Philadelphia and at the University of Pennsylvania, graduating from the College Department in 1873, and from the Medical School in 1876. His graduation thesis was on "Foreign Bodies in the Brain." He then became an intern for two years in the newly opened University Hospital, serving first under D. Hayes Agnew and then under John Ashhurst, Jr. Agnew was the professor of surgery in the University, while Ashhurst succeeded Neill in the chair of clinical surgery in 1877. Both were strong men and outstanding figures in American surgery. Agnew was the brilliant operator and popular practitioner, Ashhurst the profound student and great encyclopedist. Both interested themselves in young Wharton, and Ashhurst invited him to become his assistant in the hospital. After serving a further term as resident physician at the Germantown Hospital, he entered on his work as an assistant in the Ashhurst Clinic and assistant surgeon to the University Hospital, the duties of which position he shared for years with his close friend, Richard H. Harte. He also entered private practice at this time, and was soon appointed surgeon to the Children's Hospital, where Ashhurst was the senior surgeon.

His success in practice was assured. He had natural talents

of a high order, was extremely industrious, very conscientious and painstaking in his work, and was greatly liked for his geniality, simplicity and modesty. His patients loved him, and he was the favorite consultant and close friend of an influential group of physicians who controlled much of the practice among the old families of Philadelphia. His connection with the Children's Hospital, and later with the Mary J. Drexel Hospital for Children, gave him unusual opportunities for the study of pediatric surgery. Many of Wharton's contributions to the literature were on subjects dealing with the surgery of childhood. He was the outstanding tracheotomist in Philadelphia before the antitoxin era, when diphtheria levelled a heavy toll on the juvenile population. He quickly took up intubation after Joseph O'Dwyer earned undying fame by its introduction, and for years he stood almost alone as the intubationist in private practice in the city.

In 1891 he was elected surgeon to the Presbyterian Hospital, and from then until his retirement from practice he was intimately identified with that institution and for a long time was president of its staff. At various times he held other hospital appointments in addition to those named, serving on the staffs of the Methodist and the Bryn Mawr Hospitals, the Pennsylvania Institution for the Deaf and Dumb, and as consulting surgeon to the St. Christopher's Hospital for Children. He was for many years surgeon to the Girard College, a famous and unique charitable institution and home for orphan boys, founded under the will of the old mariner, merchant and financier, Stephen Girard, and most admirably administered as a city trust.

Wharton taught at the University until 1900, serving as demonstrator of surgery, and delivering the didactic lectures on surgery for two years, during the final illness of Ashhurst. Later he was professor of clinical surgery at the Woman's Medical College. He was an active member of the College of Physicians of Philadelphia and the Academy of Surgery. Frequently a contributor to their programs, he served several terms on the council of the College and was a president of the Academy. He wrote a *Textbook of Minor Surgery* which was deservedly popular and which ran through nine editions. He collaborated as senior author with B. Farquahar Curtis, of New York, in writing a *Practise of Surgery* which had three editions and was an excellent work and full of sound practical surgical teaching. He was also a contributor to

Ashhurst's *International Encyclopedia of Surgery*, and to Starr's *American Text-book of the Diseases of Children.*

Dr. Wharton was an excellent diagnostician and a skilled therapeutist. He was a painstaking and deliberate operator, and extremely careful and assiduous in his after-treatment. He excelled in the treatment of fractures, which subject interested him greatly, and in diseases of the bones and joints. He was notably successful in the operations for harelip and cleft palate. Mention has been made of his skill as tracheotomist and intubator. His writings were sufficiently numerous for so busy a man, and were marked by thorough study of his subject, excellent judgment, broad experience and sound common sense. He was withal the most modest of men.

No memoir of Henry Wharton, or Harry, as he was known to his many friends, would be complete without some mention of his favorite recreations. These included gardening, shooting and fishing. Like many other Fellows of this Association he was an enthusiastic sportsman. The author of this sketch was for long his assistant and then his colleague. It was his privilege, besides sharing with him the tribulations of practice, to be his companion on many happy days in the field and on the water. No keener sportsman or more genial companion could be desired. He was a good shot, and for many years the Eagle Island Gun Club was his Mecca, and in the ducking season he was always scanning the sky and speculating on the prospects for sport. He was also a popular member of the Philadelphia and University Clubs and of the Medical Dining Club, and with his strongly developed social instincts and his keen sense of humor he delighted in these enlivening contacts with his friends.

Dr. Wharton married Edith R. Booth, of New Castle, Delaware, who with five children survives him. He died, after a lingering illness, in Philadelphia, December 3, 1925.

J. H. J.

JOHN WHEELOCK ELLIOT, M.D.

JOHN WHEELOCK ELLIOT, A.M., M.D.

1852–1925

John Wheelock Elliot was born in Keene, New Hampshire, in October, 1852. He fitted for Harvard at Phillips Exeter Academy, took his degree of A.B. at Harvard in 1874 and his M.D. four years later. He might have received his degree in 1877, but he was a house pupil at that time at the Massachusetts General Hospital and it was a hospital rule that no student should have any more dignified title than "Mister" while he was an intern.

He started his surgical career at a most fortunate period. Dr. Samuel Cabot, Dr. Henry J. Bigelow, Dr. Richard M. Hodges, Dr. Charles B. Porter and Dr. John C. Warren were the visiting surgeons, and must have been a great inspiration to any young man. Lister's work was just beginning to be recognized, and the first operation performed at the hospital under his technic was done while Dr. Elliot was a house pupil. A few months later Professor Lister himself came to the hospital and demonstrated his methods to the surgeons and house officers. After graduation Dr. Elliot went abroad, studying for two years in Vienna, Dresden, Berlin, Paris and London.

He began practice in Boston in 1881, and in addition to his private work he was on the staff of the Free Hospital for Women and the Boston Dispensary. In 1887 he was appointed to the staff of the Massachusetts General Hospital, moving up through successive stages until, in 1894, he was visiting surgeon. This position he held until 1906, when he was appointed to the Board of Consultation, a position which he held at the time of his death. Dr. Elliot's associates on the staff of the Massachusetts General Hospital were Dr. John Homans, Dr. Arthur T. Cabot, Dr. Maurice H. Richardson, Dr. Francis B. Harrington, Dr. Samuel J. Mixter and others. Surely a distinguished company, each so

different, yet each so excellent in his way. It was this group which developed the clean surgery of today at the Massachusetts General Hospital.

Dr. Elliot's contributions to the hospital and to surgery were many: When he first went on the staff the old sea-sponges were still used, and it is easy to imagine his opinion of them. He set about devising a clean substitute, and due to his efforts, through successive stages the gauze sponges were evolved. He was the first of the surgeons to systematically use gloves in operating, sometimes rubber and sometimes cotton.

He used to say he was glad he knew no anatomy, for if he did he should always be afraid of cutting something. Few knew it better as far as really important structures went, and this remark is as characteristic as anything of his surgery. He had a way of passing over unimportant symptoms and getting straight at what was the real trouble with a patient which few men could equal. Having found the trouble, he was equally rapid in making up his mind what to do and doing it.

He never was a great writer, but we who knew him at the hospital know that the cases he has reported are only a very small part of his really remarkable results. In these days of specialization it seems astounding that one man should be reporting cases of mesenteric thrombosis, laminectomy, nephrectomy and so on, but he did whatever came on his service and did it well. He had "surgical instinct" to a very unusual degree.

At the time of the World War he had given up active practice, but when the Surgical Dressing Committee needed help and advice he brought to their aid all that wealth of knowledge and good common sense which was such an encouragement to them in those dark days. In speaking at the unveiling of the tablet at the Peter Bent Brigham Hospital to the Surgical Dressings Committee, Dr. Elliot modestly spoke of viewing the work from the side lines. As chairman of the advisory committee of surgeons, his duties were manifold and his interest unflagging as long as the work lasted. His friend, classmate and fellow house-pupil, Dr. James J. Minot, has written of him, "He was true to his patients, his friends and himself, broadminded and unprejudiced."

F. G. B.

JOHN T. BOTTOMLEY, M.D.

JOHN T. BOTTOMLEY, A.B., M.D., LL.D.

1869–1925

John T. Bottomley died suddenly of heart disease in Boston on December 17, 1925. He was engaged in active surgical practice up to the day of his death. For the last two years of his life he had been forced to limit somewhat his boundless surgical activity on account of grave warnings of cardiac weakness. To his colleagues and patients he continued to show the same sunny, energetic and forceful nature which had always characterized him. His physical disability might limit the amount of his work, but it did not alter its character. He was a man of high ideals and spotless character, with a ready wit, kindly humor, great courage and strength. He radiated good will, sincerity and cheerfulness all about him.

His professional career was a distinguished one. He was born at Lee, Massachusetts, September 24, 1869. He graduated from Holy Cross College in 1889, and from the Harvard Medical School in 1894. He served as surgical intern at the Boston City Hospital, and later on its surgical staff as assistant visiting surgeon. In 1903 he went with his close friend and chief, the late John C. Munro, to the Carney Hospital, and after that brilliant surgeon's death, in 1910, he succeeded him as surgeon-in-chief, a position held to the time of his own death. In the Spanish War he served with distinction as surgeon on the hospital ship, *Bay State*. In the World War he rendered important home service as a captain in the Medical Corps.

He was elected a Fellow of the American Surgical Association in 1913. He was a fellow of the American College of Surgeons, member of the International Surgical Society, Society of Clinical Surgery, American Medical Association, Massachusetts Medical Society, New England Surgical Society and the Boston Surgical Society. Of the latter he had served as president.

It was as a clinical and consulting surgeon that he made his mark in the community. His good judgment, resourcefulness and skill were eagerly sought for all over New England. He was consulting surgeon on the staffs of no less than twelve New England hospitals. When he once took on a case he gave unstintingly of all his resources to its successful prosecution; time, money and personal comfort were of no consequence. It is no wonder that his consultants and patients showed devotion and gratitude to an extraordinary degree.

He was a well-grounded general surgeon of a fast-vanishing type. While well equipped and proficient in general surgery, his special interest lay in the surgery of the abdomen. He contributed many articles on the latter to medical literature, especially on ureteral, gall-bladder and stomach surgery. He was a diligent reader of surgical literature and extraordinarily well posted on all surgical progress at home and abroad. He had a fine taste in literature and art, and was an enthusiastic and discriminating bibliophile. He was loyal and devoted to his own religious faith, but withal broadminded and tolerant toward that of others.

His home life was very happy. He married Miss Mary A. Kenney, of Boston, in 1908, by whom he is survived as well as by five children.

Such are some of the bare facts in the life of John Bottomley, a fine, whole-souled, true American, whose life brought joy, comfort and relief to countless hundreds, and whose passing leaves a void in the lives of his friends which cannot be filled.

L. D.

E. H. BRADFORD, M.D.

EDWARD H. BRADFORD, A.B., M.D.

1848–1926

In attempting a notice of the late Dr. Edward H. Bradford, one's first feeling is one of dismay at the inadequacy of such language as one has at command to portray his character. The living, breathing man can only be known to those who were privileged to be his friends, and no recital of what he accomplished can make the picture of him complete. Yet upon this means chiefly must one depend in such an account as this. Fortunately his accomplishments best portray his character.

Able he was, and the founder of the great Boston school of orthopedic surgery; loyal and devoted to medical education and to his Alma Mater, the Harvard Medical School, which he served with distinction as dean and professor of orthopedic surgery; kind and compassionate, with Dr. Augustus Thorndike he established the Industrial School for Crippled and Deformed Children. At his death he was a director of the Massachusetts Hospital School, a similar institution, in Canton, Mass. He was an overseer of Harvard College. Such are a few of his principal activities. To all of them he devoted the enthusiastic, loyal devotion of a rare mind and a kind heart in a sound body.

His intelligent judgment of young men was one of his remarkable characteristics. In the choice of his associates and assistants, in which he had much important work to do in connection with the Harvard Medical School and his various hospitals, his first loyalty was to the institutions. He considered character and ability before social position and friendly association. No one was more loyal to his friends than he, but the school or the hospital stood first. The speed and accuracy with which his mind leaped to right conclusions was remarkable. To younger men who were fortunate enough to become his associates he was more than generous. He helped them to opportunities for practice, gave encouragement and advice, and, above all, stimulated them by the example of his own contagious enthusiasm and hard work.

Himself always at his best, he brought out the best in others, nor was it strange that all who knew him loved him. The institutions that he founded and upon which he stamped his character are his greatest monument, and of these those who mourn for him may well be proud.

In the latter years of his life he bore the infirmities caused by a serious accident, resulting in the gradual invasion of blindness (which became almost complete) with a courage and cheerfulness which his friends had never seen surpassed, and when certain avenues of activity were cut off he entered upon others with the enthusiasm of a boy.

In history, his interest was great, especially in the Civil War, his knowledge of which was worthy of a historian. Of this he loved to talk among his friends. Toward the end of his life he was able to give more time to the study of the Greek and Roman classics, in which his interest had always been great. In social life, and with fellow club members he was all things to all men, meeting all his friends on their own ground, with sympathetic interest.

A few brief facts about his life: He was a lineal descendant of Governor William Bradford, who came over in the *Mayflower*, and John Alden, of the Plymouth Colony, was also an ancestor of his. He was, however, not one of those whose fame depends upon illustrious ancestry alone; far from it. He was born in Boston in 1848; graduated from Harvard College in 1869, receiving the degrees of A.M. and M.D. from Harvard in 1872; was an intern at the Massachusetts General Hospital, and studied abroad a year. He began his association with the Children's Hospital immediately on his return, and was for fourteen years a surgeon at the Boston City Hospital, where he had a wide surgical experience, which was of great help to him in his subsequent work in the field of orthopedics. For thirty-eight years he was on the faculty of the Harvard Medical School, beginning in 1881 as clinical instructor in orthopedic surgery. In this department he rose to be the first professor of orthopedic surgery. His position in his chosen field was recognized by his election to the presidency of the American Orthopedic Association. He was married in 1900 to Edith Fiske, who bore him three sons and a daughter, who were the pride and joy of his later life, and who have all distinguished themselves in athletics and scholarship.

In orthopedic surgery he was a pioneer, and spent his life in the constant joy of contributing to the remarkable progress in that field. A few of the subjects to which he contributed in this department of surgery may be mentioned. In congenital dislocation of the hip he was one of the earliest and most effective workers. In tuberculosis of the hip and spine he also did pioneer work. In fact, there is hardly a subject in the field of orthopedic surgery upon which he had not thought and written. His *Textbook of Orthopedic Surgery*, written in conjunction with the late Dr. Robert W. Lovett, has for many years been the leading textbook on that subject. His establishment of the schools for crippled children, the first in this country, was remarkable. Now they are found all over the land, but, says Dr. Augustus Thorndike, in a notice of his death, "Had it not been for Dr. Bradford's idealism, there might not have been any schools at all." He had the vision, and so effective was his personality, so transparent his honesty, and so great his enthusiasm, that he found the means and the co-workers to make his dreams come true. He was indeed no narrow specialist, and looked at each individual as a human being and not as an orthopedic case. Kind, keen and accurate was his mind, and great his character and accomplishment. He embodied in his life the character of "the happy warrior."

I cannot close this brief notice more fittingly than by reproducing a note his son found after his death, apparently as a preliminary to an essay on medical education, which will illustrate the breadth and sanity of his views on that subject, and happen to embody, as my own words could not do, the ideals which he expressed in his own life. The note is as follows: "The doctor should be broadly human. He must deal with the vagaries of age and the fancies of youth, the sports of boys and the appetites of men. In his profession he tests the aviator and rations the soldier, estimates the endurance of the laborer, cares for the worried mother and relieves the desk-ridden financier. His thought must reach to the ideals of the clergyman and interpret the flesh-prompted dreams of the man of the world. And in this service, neither the precision of science nor the efficiency of business methods will suffice; for above all else the practitioner must preserve and exercise the kindly indulgence of a considerate friend. In what academy can these lessons be taught?"

F. B. L.

EDWARD W. WALKER, M.D.

EDWARD WOOD WALKER, A.B., M.D.

1853–1925

Born, September 3, 1853, a son of Judge Timothy and Ellen Page (Wood) Walker.

He graduated from Harvard in 1874 with an A.B. degree. His M.D. degree was obtained from the Medical College of Ohio in 1878.

Dr. Walker was appointed pathologist to the Cincinnati Hospital in 1880, and in 1882 was appointed surgeon to the same institution. He was appointed professor of the principles and practice of surgery in the Miami Medical College in 1886. In addition to these appointments he held the position of surgeon to St. Mary's Hospital, Episcopal Hospital for Children and to the Deaconess' Hospital. Dr. Walker also served his city as a member of the Board of Health from 1909 to the time of his death.

Dr. Walker was a member of the American Surgical Association from 1891 to the time of his death.

He married Miss Inez Hatfield in 1897. They had no children.

Dr. Walker has been an exceedingly useful man in his professional capacity as well as in the discharge of his duties as a citizen. As a physician and a teacher he will be long remembered as an honest, modest practitioner of surgery—a safe consultant and an expert operator. His teaching was conservative and very thoughtful. His manner of teaching was not forceful, but his students were all impressed with his evident honesty and clearheadedness. They grasped the ideas he sought to convey and profited thereby. Because of his modesty and clear thinking he became a great favorite with the students.

Dr. Walker was of a very retiring disposition, very kind and considerate in his dealings with everybody, loyal and true to the best ideals of his profession, and a stubborn foe to every influence which tended to belittle or besmirch the profession of medicine.

His writings, while not voluminous, were always characterized by their clearness of thought and clarity of expression.

During the past few years Dr. Walker had gradually withdrawn from active practice and from attendance upon medical meetings, but there were many of his patients who insisted on having his care so long as he was physically able to help them bear their burdens.

He died, October 7, 1925, in his seventy-third year.

J. C. O.

RICHARD H. HARTE, M.D.

RICHARD H. HARTE, M.D., C.M.G., Hon. F.R.C.S. (Ire.)

1855–1925

Richard Hickman Harte was born at Rock Island, Illinois, October 23, 1855, passed his entire professional life in Philadelphia and died, November 14, 1925, at Vicksburg, Mississippi.

He graduated in 1878 from the Medical Department of the University of Pennsylvania, and received his early training in surgery in the University Hospital, as assistant to Agnew and to Ashhurst; and later in the Pennsylvania Hospital, where he became a surgical chief in 1893. He also served as surgeon to the Episcopal Hospital (1889–1904), to St. Mary's Hospital (1893–1899) and to the Orthopædic Hospital (1904–1914).

Possessed of an ample fortune, Dr. Harte may be said to have practised his profession as Johann Sebastian Bach wrote music, "for the glory of God and for a pleasant occupation." He never had a very large private practice, but delighted in his work in the hospital wards, paying particular attention to the old, the helpless and the miserable, especially to those unfortunates whose sojourn is long in the dreary dwellings which border on the shades of death. Though never of very robust physique himself, he radiated an atmosphere of cheerfulness and hope among his patients, who cherished his visits and appreciated his neatness and gentleness in dressing their wounds more than his operative skill, of which they knew nothing.

Between the ages of fifty and sixty years, Dr. Harte gradually withdrew from practice and resigned, one after another, all his hospital appointments except that of surgeon to the Pennsylvania Hospital.

Elected a Fellow of this Association in 1895, he soon became an active and interested member, rarely missing the annual meetings and contributing a number of valuable papers to its *Transactions*. From 1900 to 1909 he served as Recorder, until his election as President in 1910.

Of Dr. Harte's war service it is impossible to speak adequately in this place. Feeling very strongly the call of duty to assist the Allies against their enemies, he left his home and his many engagements in this country early in 1916, and served for many months in the American Hospital at Neuilly-sur-Seine (Paris). Returning to Philadelphia in the autumn of 1916, and foreseeing the entrance of the United States into the conflict, he set about organizing two base hospitals, one in connection with the Pennsylvania Hospital (which became Base Hospital No. 10, A. E. F.); the other in connection with the Episcopal Hospital (which became Base Hospital No. 34, A. E. F.). Leaving, as its director, with Base Hospital No. 10 for France in the spring of 1917, he remained on active duty in France until after the armistice. During his absence he had the greatest sorrow of his life, the death of his wife; but he sought to forget his grief in constant activity, saying that he knew it would be her wish for him to complete the task he had undertaken, and he never faltered. He was rapidly promoted to the rank of colonel, and illness alone prevented him on his return to this country in the winter of 1918 from serving as chief surgeon of the Walter Reed General Hospital, Washington, D. C. He received from General Pershing a citation for "exceptionally meritorious and conspicuous service." His work with the British Army was of such importance that it was mentioned in dispatches by General Haig, and later he was made a companion in the British order of St. Michael and St. George. The King of Belgium decorated him as companion in the Order of Leopold and he was made honorary fellow of the Royal College of Surgeons of Ireland for "conspicuous service rendered to the British Expeditionary Forces." From our own country he received the Distinguished Service Medal.

Dr. Harte wrote very little. He took special interest in the surgical complications of typhoid fever, and published a number of important papers on perforation of and on hemorrhage from the intestines during that disease. He had probably the largest personal experience of such complications of any surgeon in the world.

Dr. Harte had a rare intuition of diagnosis and prognosis, a surgical judgment which was almost infallible and he operated with an ease and deftness which I have never seen equalled either in this country or abroad. Every scalpel that he used seemed

sharp, tissues fell asunder beneath its strokes as if by magic and with nearly complete absence of bleeding; ligatures dropped from his fingers as if already tied, and his wounds healed with the most surprising rapidity and with minimum scarring. It was with the deepest regret that his assistants saw him abandon his career as operating surgeon so comparatively early in life. He resumed it only for a short time in France, during the war, and was delighted that the first patient on whom he operated at the front (a soldier with multiple gunshot perforations of the bowel) made an uneventful recovery.

He was, as I have said, never robust. Subject to bronchial inflammations, he rarely passed a winter without being laid up in bed for a few days on one or more cccasions. He also suffered a good deal from a stiff and painful right shoulder, which he called "neuritis," and which made him miserable sometimes for weeks at a time. When run down, nothing would restore his health so soon as a river trip, or coasting expedition, either in his own yacht or in that of his bosom friend, Dr. William J. Mayo.

Leaving Philadelphia with a bad cold and with his arm in a sling, on November 2, 1925, he joined Dr. Mayo on his boat on the Mississippi River two days later at Memphis; had a severe chill and took at once to his bed. As soon as the presence of pneumonia was suspected, he was transferred to the hospital at Vicksburg, and there, on the shores of his favorite river, after a brave fight against the disease for more than a week, attended by Dr. Mayo and other friends, and with his children present, his spirit passed on to the other shore of the river of death. He had lived his life with a conscience void of offense toward God and toward man.

A. P. C. A.

ANTOINE DEPAGE, M.D.

ANTOINE DEPAGE, Hon F.R.C.S. (Eng.)

His death, June 9, 1925, terminated the wonderful career of a very great man. His entire life represented service and achievement; it may be divided into three periods:

The first represents the steady progress of a young man to the position of Belgium's outstanding surgeon. Always a big man, he was also a strong man, the leader and inspiring example. In this period he developed his genius for organization, particularly in hospital construction and administration. In his splendid private hospital he founded the first training school for nurses in Belgium; Edith Cavell was its head. With his splendid Belgian colleagues he founded the International Surgical Association. As secretary, 1905–1911, president, 1911–1914, he was its great driving force.

The second period dates from his organization and conduct of the Turkish Red Cross in the Balkan War of 1912, at a great sacrifice of his time and resources. His great judgment foresaw the aftermath of this struggle and his presidential address in 1914 was on military surgery of the future. A few weeks later he was destined to blaze the way as much or more than any other living being.

It was his glorious conception of bringing the well-equipped hospital to the wounded soldier that revolutionized treatment. Beginning with nothing, there was established at La Panne, seven miles from the front, not solely a hospital, but in reality a clinic for war surgery, complete in every detail, even to well-organized laboratories for the physiologic investigation of shock. By 1916 its importance had become such that the allied armies sent countless observers to learn the lessons of progress and research.

The third, or post-war period, saw Depage continuing with all his energies the task of not only salvaging the wrecks, but also making this a better world to live in. He brought about a complete rehabilitation of the Medical Department of the University

of Brussels, receiving the generous assistance of the Rockefeller Foundation, which instantly recognized the worth of the cause and its faith in Depage. He instituted farsighted measures for public health in Belgium; he developed an active crusade for the investigation and treatment of cancer.

An organizer of the League of Red Cross Societies, he modestly declined to become its president. He was elected a member of the Belgian senate, to which he gave the utmost of his efforts to improve the condition of the people. He had to an extraordinary degree the confidence, support and affection of the King and Queen of the Belgians.

Depage had a splendid personality. Big and handsome, a somewhat brusque manner really was a cloak for real diffidence. He had the beautiful and true faith in his purposes, without which even the best qualified may fail.

To him came one of the greatest sorrows: The beautiful and gifted Marie Depage died in the *Lusitania* torpedoing, returning from a successful mission to the United States. His later years were made hard by a series of serious illnesses, succumbing finally after a third operation.

It is gratifying to think that during his lifetime he received recognition of his services. To indicate even partially some of these would exhaust the limit of this observation. From Belgium and France every possible honor and decoration—honorary membership in the Institut de France, and the presidency of the French Surgical Association. From England the Order of the Bath and, particularly gratifying, the Distinguished Service medal of the United States.

He is survived by three splendid sons. There is also a fine grandson, in whom we shall always have a particular interest as he was born during the two years' residence of Dr. and Mrs. Pierre Depage at Rochester, Minn.

C. L. G.

LUCIUS W. HOTCHKISS, M.D.

LUCIUS WALES HOTCHKISS, A.B., M.D.

1859–1926

THE death of Dr. Lucius Wales Hotchkiss at his home in Santa Barbara, California, on April 11, 1926, brought much sorrow to a very large group of friends. His genial disposition and his square-going and right-minded activities had endeared him to an unusual number of devoted friends and acquaintances.

He was born in New Haven, Connecticut, in 1859; received his precollegiate education in Brooklyn and was graduated from Columbia University in New York City in 1881, with the degree of B.A., and from the College of Physicians and Surgeons, the medical department of that university, in 1884. His intern service was on the second surgical division of Bellevue Hospital, in New York City.

His further surgical training was most comprehensive. His activities were divided among several institutions, a custom which was more common thirty-five years ago than now, since the individual surgical service in many institutions comprised only three months in each year. At Bellevue Hospital he was assistant surgeon during twelve years, attending surgeon for ten years and consulting surgeon ten years. At the Roosevelt Hospital he worked in the out-patient department nine years, was assistant to the attending surgeon three years, an attending surgeon during ten years and consulting surgeon seven years. At the Colored Hospital (now the Lincoln Hospital) he was attending surgeon five years. At the J. Hood Wright Hospital (now the Knickerbocker Hospital) he was attending surgeon thirteen years and consulting surgeon sixteen years.

At the College of Physicians and Surgeons he was assistant demonstrator of anatomy from 1890 to 1897, and at a later time was instructor in surgery and then professor of clinical surgery in that institution. He was also professor of anatomy in the

Women's Medical College of the New York Infirmary for ten years.

He was also much interested in the work of surgical and medical societies, especially the New York Surgical Society, of which he was president, 1901 to 1903; the Bellevue Alumni Association, of which he was president for the fiscal year 1896–1897; the Academy of Medicine; the International Surgical Society and the Society of Clinical Surgery.

It is easily seen that these many activities filled up several years of his busy life.

He was always a careful clinician, spending his days in painstaking and skilful attention to the many patients who came to him in his private practice and in his institution work.

He made many valuable contributions to surgical literature. One of the most important was in the development of the problem of appendicitis. As we now remember the early operations for appendicitis we wonder at their complexity; there was much handling and sponging of the intestines, considerable irrigation and extensive drainage. Dr. Hotchkiss early saw that much harm could come from these procedures and he early appreciated the patient's inherent power of controlling peritoneal sepsis. With these elements in view, he operated upon a series of cases of acute appendicitis, using the minimum of trauma and drainage; his results were so good as to mark a distinct stage in the development of the subject. He also made very valuable contributions to the method of operating on sliding hernia.

In the index of the *Annals of Surgery* we find reference to about one hundred and twenty contributions which he had made there, most of them reports concerning the serious and noteworthy cases or groups of cases which had come under his care in his hospital work.

He was always fond of outdoor life, and in 1922 he moved from New York City to Santa Barbara, California. He quickly built up a surgical clientele there and was in active practice at the time of his death.

Mrs. Hotchkiss, a son, Henry G. Hotchkiss, who is practising law in New York City, and three daughters survive him.

C. N. D.

GABRIEL MAUNOURY, M.D.

GABRIEL MAUNOURY

1850–1925

Gabriel Maunoury died at Chartres, December, 1925, aged seventy-five years. He belonged to one of the fine old provincial families who have been the backbone of France, and was a brother to the heroic Marechal Maunoury, a great factor in the victory of the Marne.

After brilliant medical studies and internship he continued his studies in other countries, particularly with Lister, and was head of a Red Cross unit in the Turko-Russian War.

In 1878 he settled in his home town, succeeding his father as surgeon to the hospital. He soon created a modern and model service and his successful work attracted widespread attention.

During the World War he again became an army surgeon in charge of two military hospitals.

He was the local head and organizer of many welfare movements. Despite his many responsibilities he served several terms as a member of the French Parliament.

As president of the French Surgical Association in 1918 he issued most cordial invitations to the members of this Association to take part.

As an Honorary Fellow he was particularly gratified to be present at the ceremony of laying a wreath on Pasteur's tomb in behalf of the American Surgical Association.

C. L. G.

JAMES M. BARTON, M.D.

JAMES M. BARTON

1846–1926

James M. Barton was born in Philadelphia October 16, 1846. He was educated in the schools of Philadelphia and studied medicine in the Jefferson Medical College, from which he graduated in 1868.

In his student days he was taught by many eminent men, including Samuel D. Gross, the elder Pancoast, Dixon, J. M. DaCosta (who was then a lecturer on clinical medicine) and James Aitken Meigs. Soon after his graduation he became a clinical assistant on the clinics of Gross and Pancoast, and also a quizmaster on surgery in the college. He attained the position of chief of Dr. Gross's Clinic. If we compare medical with military matters, we may say that to be chief of Dr. Gross's Clinic at that time was about equivalent to the position of Berthier as chief of staff of the great Napoleon, because Gross, at that time, was called the "Emperor of American Surgery."

A likeness of Barton when he was chief of Dr. Gross's Clinic can be identified in the celebrated picture of Dr. Gross operating, a picture painted by Eakins, which hangs in the Jefferson Medical College and which is regarded by artists as a masterpiece. Dr. Barton is shown assisting in the operation and picking up blood-vessels by means of a tenaculum, the slow method that was in use until the introduction of the hemostatic forceps.

Some years before Dr. Gross's retirement from the chair, in 1882, Dr. Barton retired from the position of chief assistant and became surgeon to the German Hospital. Later he was made surgeon to the Philadelphia Hospital, and in 1882 he was made surgeon to the Hospital of the Jefferson Medical College, which at that time had four surgeons and the professors of surgery.

In 1898 Dr. Barton was made clinical professor of surgery. Among his colleagues in the Jefferson Medical College Hospital

were Samuel W. Gross, William S. Forbes, John H. Brinton, Charles B. Nancrede, R. J. Levis, W. J. Hearn, Frank Maury and W. W. Keen. He outlived all of this distinguished group with the single exception of the venerable Professor Keen.

Dr. Barton was always interested in the surgery of the abdomen, although he was a general surgeon in the true sense of the term. He was one of the earliest of surgeons to become convinced of the reality of the views of Reginald Fitz on appendicitis and was a strong believer in operation at a time when many hesitated and doubted. He was an excellent teacher, was always clear and concise, but made no attempt at eloquence. He had a solid ground-work of anatomy, was a good diagnostician, formed his judgments rapidly, operated dextrously, was bold and always careful. He was a loyal and lovable friend and were he your advocate in a contest you were never obliged to visit him to take the temperature of his support. He never played to the galleries. He was, as a general thing, a rather reserved man, but often was outspoken in his opinions of men and things. Because of this tendency he naturally made enemies, but the enemies he made in this way were compliments to his honesty. When things went wrong in an operation he was capable of whiffs of irritability in which he used language emphatic and sometimes picturesque. He wrote little, but what he did write always contained sound surgery. For many years he regularly attended the meetings of societies, occasionally read papers and frequently participated in debates. He did a vast number of surgical operations. His real field was in the hospital rather than in the laboratory or the library. A great deal of his work was in the country districts of Pennsylvania, New Jersey and Delaware, exhausting work, as all of us have found it to be who have gone through the experience.

A severe attack of grippe led him to resign his position as clinical professor of surgery at the end of the year 1899. He went to Atlantic City and remained there for the balance of his days, happy with his gun, his dogs, his boat and his rod. It was said that no one knew the waterways around and about Atlantic City better than Barton.

He died June 28, 1926, at the age of eighty. His life was long, happy and useful.

J. C. DaC.

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